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DISEASES OF THE NOSE-MOUTH-THROAT AND LARYNX

BY

ALFRED BRUCK M.D.

TRANSLATION SUPERVISED AND EDITED

BY

F. W. FORBES ROSS, M.D., F.R.C.S. ENG.

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AUTHOR'S PREFACE.

This book is intended to meet the requirements of the men in general practice. Relying on the long experience in my own special practice, I have tried to take the standpoint of the practitioner. Discussion of the theoretically important questions cannot be entirely omitted from a treatise like this. Consequently the large amount of material at my command has been condensed, and references to other authors have been restricted to such as could not be neglected. Much space has been given to the GENERAL SECTION in each of the four parts of the book. Anatomical and physiological points must of necessity receive attention in a text-book, as they are needed for a proper understanding of the pathological process, viz., accessory cavities or nervous lesions. Still, I have endeavored to confine myself to the essential points, so as not to weary the reader. Full allowance has been made to the methods of examination. These are well illustrated by diagrams, partially schematic. For a clearer understanding of the operative technique I have, wherever practicable, shown the instruments *in situ*. In order to achieve clearness and systematic arrangement, the book has been divided into FOUR PARTS.

I trust that the book will be favourably received by my colleagues among the specialists, and prove a useful addition to the library of the General Practitioner. *Habent sua fata libelli.*

THE AUTHOR.

BERLIN.

TRANSLATORS' PREFACE.

The Translators of this useful book for Practicians and Students, in the course of their labours were impressed with the fact that the Original Author had missed nothing which was of real practical value, and at the same time had been able to get so much into so small a compass. One of the most salient features of the work is the prevailing absence of any unnecessary verbiage or "padding out"—in order to produce a lengthy and imposing work. This has been successfully achieved in the smallest possible space compatible with real practical value. The Translators have endeavored as much as possible to closely follow the German text; even at the risk of not producing "classical English" at the expense of the exact "shade of meaning" or sense which the author originally endeavored to convey.

F. W. Forbes Ross, M.D., F.R.C.S. England.
Friedrich Gans, M.D.

London.

TABLE OF CONTENTS.

PART I.

DISEASES OF THE NOSE AND ITS ACCESSORY CAVITIES.

General Section.

PAGE

Anatomy 1
  The External Nose 1
  The Internal Nose 2
  Accessory Nasal Cavities 6
  The Mucous Membrane of the Nose 10
    Blood Vessels 11
    Lymph Vessels 12
    Nerves 12

Physiology 13

Methods of Examination 16
  The Examination from in Front 16
    Probing (Examination by the Probe; Sounding) 21
    Anaesthesia and Artificial Anaemia (Anaemization) 21
    Deflation of the Nose 23
  Posterior Rhinoscopy (*Examination from Behind*) 24
  Transillumination 30

Routine of Examination 31
  Anamnesis 31
    Heredity—General Conditions of Life—Previous Diseases—Present Diseases—Subjective Symptoms—(*a*) Pain—(*b*) Psychic Disturbance—(*c*) Obstruction—(*d*) Sneezing—(*e*) Disturbances of the Sense of Smell 31
  Status Praesens 32
    External Nose—Inspection—Palpation 32
    Nasal Cavity—(*A*) Examination from in Front—(*a*) Test of Air-permeability—1. Stenosis-bruit—2. Mouth-breathing—3. Alteration of voice—4. Disturbance of speech—(*b*) Ascertaining of Nasal Foetor—(*c*) Anterior and Middle Rhinoscopy—Quantitative Changes—Qualitative Changes—(*d*) Examination of the Sense of Smell 33

PAGE
(*B*) Examination from Behind—Posterior Rhinoscopy—Palpation of the Naso-pharyngeal Space.................................... 34
(*C*) Transillumination—1. The Maxillary Sinus—2. Frontal Sinus.......................... 35
Other Organs.. 35
1. Eye—2. Ear—3. Upper Air-passages—4. Teeth—5. Brain—6. Stomach—7. Reflex Disorders......... 36
General Therapy.. 37
General Measures.. 37
Local Treatment.. 37
(*a*) Cleaning the Nose—Blowing—Wiping Out the Nose—Gottstein's Tamponade—Douching—Precautions to be taken in Douching the Nose...... 37
(*b*) Treatment by Drugs—Brushing—Massage—Instillations—Powders—"Snuffs"—Cauterization.. 40
(*c*) Operative Treatment—2. Local Anaesthesia..... 41
2. General Anaesthesia—3. Instruments........ 41
Galvano-cauterization—Electrolysis 43
Hygiene and Prophylaxis.. 44

SPECIAL SECTION.

I. Diseases of the Skin of the Nose...................... 47
Eczema Vestibuli (Eczema ad introitum nasi—Eczema of the Entrance of the Nose)...................... 47
Sycosis Vestibuli (Sycosis; Pus-Infection)............... 48
Furunculosis Vestibuli (Furunculosis; Boils of the Entrance of the Nose)............................. 49
Comedones.. 50
Seborrhoea.. 51
Congelatio Nasi (Frost-bites).......................... 51
Disturbances of Circulation of the Skin (Copper Nose; Erythema; Angeioma; Acne Rosacea; Pendulous Nose) 52
Erythema.. 52
Telangiectasis (Naevus).............................. 53
Acne Rosacea ("Blotchy Face"; Angeiectasic Erythema). 53
II. Malformations and Deformations.................... 55
Adhesions of the Nose.............................. 55
Synechiae.. 55
Atresiae.. 56
Deformation of the Septum.......................... 57
Operative Methods:
Method of Infraction.......................... 60
Resection.. 61
Injury to the Nose.. 61
Abscess of the Septum.............................. 62
Deviations, Dislocations and Fractures of the Septum.... 62

PAGE
III. Haemorrhage 62
(*A*) Bleeding into or under the Mucous Membrane 63
(*B*) Free Haemorrhage—Local Causes—General Causes ... 63

IV. Rhinitis Acuta (Coryza; "Snuffles"; "Cold in the Head") 69

V. Rhinitis Chronica 74
1. Chronic Rhinitis (Rhinitis Chronica Simplex Hypertrophica) 75
2. Rhinitis Atrophica (Ozaena) 82

IV. Special Forms of Rhinitis 89
1. Ulcus Septi Perforans (Perforating Ulcer) 89
2. Hay-fever (Bostock's Disease) 90

VII. Acute and Chronic Infectious Diseases 92
1. Acute Exanthemata 92
2. Influenza 93
3. Gonorrhoea 93
4. Rhinitis Diphtherica (Fibrinosa) 93
5. Tuberculosis and Lupus 96
6. Syphilis 99
7. Rhinoscleroma 105
8. Malleus (Glanders; Farcy) 107
9. Leprosy (Lepra; Elephantiasis Graecorum) 108

VIII. Foreign Bodies and Parasites 109
1. Foreign Bodies 109
2. Parasites 113
Actinomycosis 114

IX. Tumours 114
1. Benign New-growths 114
(*a*) Mucous Polypi 114
(*b*) Benign Tumours of Special Kind 119
2. Malignant Tumours 119

X. Nervous Lesions 121
1. Disorders of the Sense of Smell 121
(*a*) Hyposmia and Anosmia 121
(*b*) Hyperosmia. (*c*) Parosmia 123
2. Disorders of Sensibility 124
(*a*) Anaesthesia—(*b*) Hyperaesthesia—Paraesthesia .. 124
3. Nasal Reflex Neuroses 125
Intranasal Reflex Neurosis 126
Nervous Cold—Erythema of the External Nose—Influence of the Sexual Sphere on the Nose 127
Reflex Neuroses in other Organs 128
Asthma, 128—Spasmodic Cough, 129—Cardiac Neuroses, 129—Dysmenorrhoea, 129—Other Neuroses, 129.

THE DISEASES OF THE ACCESSORY CAVITIES.

PAGE
I. Inflammations 132
(*a*) General 132
(*b*) Special 137
1. Inflammation of the Antrum of Highmore 137
(*a*) In Acute Inflammation of the Maxillary Sinus, 141—(*b*) Sinusitis Maxillaris Chronica (Chronic Inflammation of the Maxillary Cavity), 143.
2. Inflammation of the Frontal Sinus (Sinusitis Frontalis) 146
(*a*) in Acute Inflammation—(*b*) Chronic Sinusitis. 150
3. Inflammation of Ethmoidal Cells (Cellulitis Ethmoidalis; Ethmoiditis) 152
4. Inflammation of the Sphenoidal Sinus (Sinusitis Sphenoidalis) 157
II. New-growths 162
Benign Tumours 162
Malignant Tumours 163
III. Injuries 164
IV. Foreign Bodies and Parasites 164
V. Tuberculosis and Syphilis 165

PART II.

DISEASES OF THE MOUTH.

General Section.

I. Anatomy 169
The Lips—The Tongue, 169—The Muscles of the Mouth, 171—(*a*) Muscles of the Lips—(*b*) Muscles of the Floor of the Mouth and of the Tongue, 171—Glands, 172—Blood-vessels, 173—Lymph-vessels, 173—Nerves, 174.
II. Physiology 174
III. Method and Course of Examination 176
(*A*) Method of Examination, 176—(*B*) The Course of Examination, 177—Disorders of Speech—Disorders of Respiration—Swallowing, 177—Anomalies of the Secretion—Increased Secretion—Paralytic Salivation—Diminution of the Salivary Secretions, 177—Disorders of Sensibility and Taste, 178.
IV. Hygiene and Prophylaxis 179

SPECIAL SECTION.

PAGE

I. **Malformations and Deformities** 180
Etiology and Pathology—(*a*) In the Lips, 180—(*b*) In the Palate, 181—(*c*) In the Tongue, 181—(*d*) In the Jaw, 181.

II. **Inflammations** 182
Acute Stomatitis (Stomatitis Acuta) 182
1. Stomatitis Acuta Catarrhalis (Acute Catarrh of the Mouth) 183
2. Phlegmonous Stomatitis 184
3. Stomatitis Exsudativa 187
    (*a*) Herpes Labialis et Buccalis (Stomatitis Herpetica; Herpes of the Lips or Cheek), 187—(*b*) Pemphigus, 187—(*c*) Aphthae (Stomatitis aphthosa seu fibrinosa; Aphthous Stomatitis), 187—(*d*) Foot and Mouth Disease (Epidemic Aphthous Stomatitis), 188.

III. **Stomatitis Chronica** 189
1. Chronic Catarrh of the Mouth 189
2. Glossitis Chronica Superficialis 190
    (*a*) Leucoplakia oris (Psoriasis sive Ichthyosis Oris; Leucoplakia; Buccal Psoriasis), 190—(*b*) Nigrities linguae (Lingua nigra; Black Tongue; Hairy Tongue), 192—(*c*) Lingua geographica (Exfoliatio Linguae Areata) (Geographical Tongue; Annulus Migrans), 193—(*d*) Moeller's Glossitis Superficialis, 194.
3. Glossitis Chronica Parenchymatosa 195

IV. **Stomatitis Ulcerosa (Chronic Ulceration of the Tongue)**. 196
(*a*) Idiopathic Ulcer—(*b*) Symptomatic Ulcer—(*c*) Traumatic Ulcer—196.
(*a*) Stomaca (Ulceration of the Mouth) 106
(*b*) Stomatitis Ulcero-Membranosa (Angina of Plaut-Vincent) 198
(*c*) Bednar's Aphthae 199
(*d*) Stomatitis Scorbutica (Scurvy) (Scharbock) 199
(*e*) Stomatitis Mercurialis (from Mercurial Poisoning) 200
(*f*) Stomatitis Ulcerosa Traumatica (Traumatic Ulcer of the Mouth) 201

V. **Stomatitis Gangraenosa (Noma; Gangrene of the Mucous Membrane; Cancrum Oris)** 202

VI. **Acute and Chronic Infections** 203
1. Acute Exanthemata 203
    Measles—Scarlatina—Fever, 203—Variola—Varicella, 204
2. Gonorrhoea 204
3. Diphtheria 204
4. Tuberculosis and Lupus 204
5. Syphilis 207
6. Malleus—Anthrax—Lepra 212

PAGE
VII. Parasites of the Mouth (Mycoses) 212
  1. Thrush 212
  2. Actinomycosis 214
  3. Other Mycoses 215
VIII. Tumours 215
  1. Tumours of the Lip 216
  2. Tumours of the Gums 217
  3. Tumours of the Tongue 217
    (*a*) Cysts, 217—(*b*) Hypertrophy of the Lingual Tonsil, 218
    Malignant Tumours 219
    Cancer of the Tongue 219
IX. Nervous Disorders 220
  1. Disorders of Taste 220
    (*a*) Ageusia and Hypogeusia, 220—(*b*) Hypergeusia, 221—(*c*) Parageusia, 221.
  2. Lesions of Sensibility 222
    (*a*) Anaesthesia—(*b*) Hyperaesthesia—(*c*) Paraesthesia, 222.
  3. Disorders of Motility 222
    (*a*) Paralysis, 222—(*b*) Spasms, 223.
X. Diseases of the Salivary Glands 224
  I. Inflammations 224
    (*a*) of the Parotid Gland, 224—(*b*) of the Submaxillary and Sublingual Gland, 226—(*c*) Pfeiffer's Glandular Fever, 226—(*d*) Angina Ludovici, 227.
  II. Salivary Calculi 228
  III. Tumours 229

PART III.

DISEASES OF THE PHARYNX.

General Section.

I. Anatomy 233
  (*a*) Pars Nasalis, 233—(*b*) Pars Oralis Pharyngis, 236—(*c*) Pars Laryngea Pharyngis, 237—Muscles of the Pharynx, 238—Vessels, 239—Nerves, 240.
II. Physiology 240
III. Method of Examination 243
  (*A*) Examination of the Naso-Pharyngeal Space 243
  (*B*) Examination of the Oral Part 243
  (*C*) Examination of the Laryngeal Part 244

PAGE
IV. Course of Examination 245
Anamnesis 245
(*a*) General Symptoms, 246—(*b*) Disorders of Sensibility, 246—(*c*) Disorders of Swallowing, 246—(*d*) Disturbances of Respiration, 247.
Status Praesens 247
(1) Pharynx—(*a*) Oral Part, 247—(*b*) Nasal Part, 247—(*c*) Laryngeal Part, 248—(2) Other Organs, 249.
V. General Treatment 249
1. General Measures 249
2. Local Treatment 250
(*a*) Applications and Fomentations, 250—(*b*) Treatment by Congestion, 251—(*c*) Treatment by Drugs, 252—(*d*) Electrical Treatment, 253—(*e*) Operative Treatment, 254.
VI. Hygiene and Prophylaxis 254

SPECIAL SECTION.

I. Malformations and Deformities 255
1. Abnormalities of Growth 255
2. Adhesions and Strictures 257
Appendix—Injuries to the Throat 259
II. Acute Pharyngitis 259
1. Acute Catarrhal Pharyngitis 260
(*a*) Retronasal Pharyngitis 261
(*b*) Acute Pharyngitis; "Sore Throat" 261
(*c*) Acute Tonsillitis 262
2. Phlegmonous Pharyngitis 265
(*a*) Tonsillar Abscess 266
(*b*) Peritonsillar Abscess 266
(*c*) Retropharyngeal Abscess 268
(*d*) Erysipelas of the Pharynx 269
(*e*) Acute Infectious Phlegmon of the Pharynx 269
3. Exudative Pharyngitis 272
(*a*) Herpetiform Pharyngitis 272
(*b*) Pemphigus 273
(*c*) Aphthae 273
(*d*) Other Exudative Processes 273
III. Chronic Pharyngitis 274
(*a*) Chronic Retronasal Catarrh 275
(*b*) Chronic Pharyngitis 276
(*c*) Lateral Pharyngitis 277
(*d*) Tonsillar Pharyngitis 277
IV. Adenoid Vegetations 283
Complications: (*a*) During Operation—(*b*) After Operation 292
V. Enlarged Tonsils 293
Complications During and After Operation 295

PAGE
VI. Acute and Chronic Infections 297
1. Acute Exanthemata 297
Measles—Scarlatina, 297—Smallpox, 298—Chicken-pox, 299.
2. Typhoid Fever 300
3. Diphtheria 300
Complications and Sequelae 304
4. Tuberculosis and Lupus 308
5. Syphilis 311
6. Scleroma 314
7. Glanders 315
8. Leprosy 316

VII. Mycoses 316
1. Thrush 316
2. Leptothrix 317
3. Other Mycoses 318

VIII. Foreign Bodies 318
Appendix—Calculi 320

IX. Tumours 321
1. Benign Growths 321
(*a*) Typical Naso-pharyngeal Polypi 323
(*b*) Retropharyngeal Goitre 326
2. Malignant Tumours 327
(*a*) Sarcoma and Carcinoma 328
(*b*) Lymphosarcoma 330

X. Nervous Disorders 331
1. Disorders of Sensibility 331
(*a*) Anaesthesia: (1) Central Causes—(2) Peripheral Causes, 331—(*b*) Hyperaesthesia, 332.
2. Disorders of Motility 333
(*a*) Paralysis 333
(*b*) Cramps (Spasms) 336

PART IV.

DISEASES OF THE LARYNX AND TRACHEA.

General Section.

I. Anatomy 341
The Cartilaginous Skeleton 341
(*a*) Cricoid Cartilage, 341—(*b*) Thyroid Cartilage, 342—(*c*) Epiglottis, 342—(*d*) Arytenoid, 343—(*e*) Sesamoid Cartilage, 343.
Ligaments of the Larynx 344
Muscles of the Larynx 348

PAGE
Nerves of the Larynx.................................. 353
Vessels of the Larynx.................................. 355
Mucous Membrane of the Larynx...................... 356
Appendix... 357

II. **Physiology**.. 358
Vigor—Timbre—Pitch of Voice........................ 360

III. **Method of Examination**........................... 363
(*A*) External Examination of the Larynx................363
(*B*) Internal Examination of the Larynx............... 364
1. Testing of Function.......................... 364
2. Internal Inspection of Larynx.................. 364
(*a*) Indirect Method........................... 364
Technic of Laryngoscopy................. 365
Difficulties During Examination............ 367
Variations of Indirect Laryngoscopy........ 373
1. Inspection of the Posterior Laryngeal Wall.............................. 374
2. Inspection of the Lateral Regions........ 376
3. Inspection of the Subglottic Region...... 377
(*b*) Direct Laryngoscopy (Autoscopy)........... 378
Direct Tracheoscopy and Bronchoscopy..... 379
Superior, Direct Bronchoscopy............. 380
Palpation of the Inside of Larynx.......... 382

IV. **Course of Examination**............................ 383
Anamnesis... 383
1. Heredity—2. General Conditions of Life—3. Previous Diseases.............................. 383
4. Present Diseases............................. 384
5. Subjective Symptoms......................... 384
Status Praesens....................................... 385
(*A*) External Examination: (1) Inspection—(2) Palpation, 385.—(*B*) Internal Examination: (1) Testing of Function, 385—(2) Internal Inspection, 388—(3) Palpation of the Interior of the Larynx, 390.—(*C*) Other Organs: (1) Nose; Mouth; Throat—(2) Lungs, 390—(3) Central Nervous System—(4) Reflex Disorders, 391.

V. **Treatment**.. 391
1. General Measures................................... 391
2. Local Treatment..................................... 394
(*a*) Fomentations—(*b*) Light Treatment—(*c*) Electricity.................................. 394
3. Instruments.. 400

VI. **Hygiene and Prophylaxis**........................... 402

SPECIAL SECTION.

I. **Malformations and Deformities**........................ 405

PAGE
1. Anomalies of Development........................... 405
2. Stenoses.. 408
   Tracheotomy... 417
   Intubation and "Catheterisation" of the Air-passages. 421
      (*a*) Intubation of the Larynx after *O'Dwyer*..... 422
      (*b*) Catheterisation of the Windpipe after *Schroetter*.................................. 426

II. Acute Laryngitis 431
1. Acute Catarrh of the Larynx........................ 431
   Dry Haemorrhagic Laryngitis........................ 434
   Subglottic Laryngitis.............................. 435
2. Phlegmonous Laryngitis 439
   Circumscribed Phlegmonous Inflammation.......... 442
   Diffuse Phlegmonous Inflammation................ 443
3. Exudative Laryngitis............................... 446

III. Chronic Laryngitis 447
(1) Chorditis tuberosa................................ 449
(2) Chorditis nodosa.................................. 449
(3) Chorditis hypertrophica superior.................. 449
(4) Chorditis hypertrophica inferior.................. 449
   Pachydermia laryngis.............................. 452
   Singer's Nodes.................................... 452
   Prolapsus ventriculi Morgagni.................... 453
   Laryngitis subglottica chronica.................. 453

IV. Perichondritis 458
(1) Perichondritis Arytenoida........................ 460
(2) Perichondritis Cricoidea......................... 460
(3) Perichondritis Thyroidea......................... 461

V. Diseases of the Joints............................... 464

VI. Acute and Chronic Infectious Diseases.............. 467
1. Acute Exanthemata................................. 467
   Measles—Scarlet Fever—Smallpox 467
2. Whooping-cough.................................... 468
3. Influenza... 469
4. Diphtheria.. 469
5. Typhoid Fever..................................... 475
6. Tuberculosis...................................... 477
   Appendix.. 498
7. Syphilis.. 501
8. Scleroma.. 508
9. Glanders.. 510
10. Leprosy.. 510

VII. Foreign Bodies and Parasites...................... 511
1. Foreign Bodies.................................... 511
   (*a*) in the Larynx—(*b*) in the Trachea and Bronchi.... 513
2. Parasites—Animal—Vegetable...................... 520

VIII. Injuries 521
(*A*) External Injuries.................................. 522

PAGE
1. Contusions........ 522
2. Wounds........ 522
3. Fractures........ 523
4. Dislocations........ 524
(*B*) Internal Injuries........ 524
1. Cauterisation and Scalding........ 524
2. Unintentional Operative Lesions........ 525
3. Haemorrhage and Rupture........ 525

IX. Tumours........ 525
(*A*) Benign Tumours........ 526
1. Fibroma........ 526
2. Papilloma........ 528
3. Cysts........ 529
4. Other benign Tumours........ 529
Intralaryngeal Operations........ 534
Extralaryngeal Operations........ 537
(*B*) Malignant Tumours........ 538
1. Sarcoma........ 538
2. Carcinoma........ 540
Tracheal Carcinoma........ 553

X. Nervous Lesions........ 553
(*A*) Disorders of Sensibility........ 553
(*a*) Anaesthesia........ 553
(*b*) Hyperaesthesia........ 554
(*c*) Paraesthesia........ 555
(*B*) Disorders of Motility........ 556
I. Paralyses and Pareses........ 556
(*a*) Central Paralysis of the Larynx........ 558
(*b*) Peripheral Paralysis of the Larynx........ 561
II. Spasms........ 577
(*A*) Laryngospasms........ 577
(*a*) Spasm of Glottis in Children........ 577
(*b*) Laryngospasm in Adults........ 581
(*c*) Laryngeal Crisis in Locomotor Ataxy.... 582
(*B*) Nervous Laryngeal Coughing........ 583
(*C*) Vertigo and Ictus Laryngis........ 585
III. Disorders of Coördination........ 586
(*A*) Phonetic Spasm of Glottis........ 586
(*B*) Inspiratory Spasm of Glottis........ 587
(*C*) Other Lesions of Coördination........ 588

XI. Affections of the Voice in Singers and Orators........ 589

APPENDIX—Meningitis Cerebrospinalis Epidemica........ 599
INDEX........ 603

LIST OF ILLUSTRATIONS.

FIG. PAGE
1. The osseous and cartilaginous skeleton of the septum nasi (viewed from the right side) (after *Toldt*)....... 3
2. The left lateral wall of the nasal cavity, showing the turbinals and nasal meatus (after *Toldt*)............ 4
3. Left lateral wall of nasal cavity (after *Toldt*)............ 5
4. Frontal view of the nasal cavity (after *Toldt*)........... 7
5. Frontal section through the posterior part of the nasal cavity and maxillary sinus........................ 9
6. Septum of the nose................................. 11
7. Reflector (after *Fränkel*)............................ 17
8. Electric headlamp (after *Kirstein*)..................... 18
9. Nasal speculum (after *Kramer-Hartmann*).............. 18
10. Anterior rhinoscopy. First position.................... 19
11. Anterior rhinoscopy. Second position................. 20
12. Speculum for middle rhinoscopy (after *Killian*).......... 21
13. Nasal forceps (after *Hartmann*)....................... 21
14. Politzer's bag with olive-shaped nozzle................. 23
15. Nasal aspirator (after *Schneerson*)..................... 23
16. Tongue depressor (after *Fränkel*)...................... 25
17. Posterior rhinoscopy represented from in front.......... 26
18. Posterior rhinoscopy schematically represented from the side.. 27
19. Palatine hook, (*a*) after *Krause;* (*b*) after *Schmidt*...... 27
20. Digital examination of the naso-pharyngeal space: Introduction of the index-finger into the patient's mouth.. 28
21. Digital examination of the naso-pharyngeal space: Palpation of the space schematically represented from the side.. 29
22. Post-rhinoscopic image.............................. 30
23. Lamps for transillumination (after *Warnecke*)........... 30
24. Nose spray....................................... 38
25. Nose douching with *Starke's* douching apparatus........ 39
26. Insufflator....................................... 40
27. Probe for cauterization.............................. 41
28. *a*, *Kuttner's* galvano-cautery, universal handle; *b*, flat burner; *c*, pointed burner........................ 43
29. Double needles for electrolysis........................ 44
30. Dilatation of the vessels on the projecting septum....... 53
31. Deviation in the lower septum towards the right......... 58
32. Cutting forceps (after *Grünwald-Struycken*)............. 59

FIG. PAGE
33. Nasal saw (*Schotz*) 59
34. Double chisel 60
35. *Heymann's* sharp cupped forceps 61
36. Position of the tampon (after *Hochenegg*) 68
37. Polypi at the anterior end of the middle concha 77
38. Polypoid and papillary hypertrophy of the end of the concha in the post-rhinitic picture 79
39. Polypus snare (after *Krause*) 80
40. *Beckmann's* nasal scissors 80
41. *Hartmann's* conchotome 81
42. Nasal air-filter (after *Mohr*) 92
43. Syphilitic saddle nose 102
44. Syphilitic saddle nose, seen by anterior rhinoscopy 102
45. Removal of a foreign body from the lower meatus 111
46. Holding child during removal of a foreign body from the nose 112
47. Removing a nasal polypus 117
48. *Lange's* choanal hook 118
49. Nasal elevator: (*a*) *Feldbausch-Roth*; (*b*) *Schmidthuisen* .. 122
50. Probing of the frontal and maxillary sinus and of the anterior ethmoidal cells (after *Hajek*) 139
51. Catheter tube for washing out the maxillary cavity (after *Hartmann*) 140
52. Syringe for the exploratory puncture of the maxillary cavity (after *M. Schmidt*) 141
53. Resection of the anterior end of the middle turbinal (diagrammatic) 142
54. Trocar for maxillary cavity 143
55. Gimlet for dental alveolus 143
56. Long punch forceps for the maxillary cavity (after *Hajek-Claus*) 145
57. Chronic empyema of the left frontal sinus 147
58. Catheter for washing out the frontal sinus (after *Killian*) .. 149
59. Exploratory opening of the frontal sinus in the eyebrow . 149
60. *Grünwald's* rotatory nasal forceps 151
61. *Hajek's* instruments for opening and scraping out the ethmoidal labyrinth 156
62. Probatory examination of the sphenoidal sinus 159
63. Revolving punch forceps for resecting anterior wall of sphenoidal sinus 161
64. Cavum oris (after *Toldt*) 170
65. Macroglossia (after *Hochenegg*) 195
66. Tuberculosis of the tongue (*Hochenegg*) 205
67. Plaques muqueuses 208
68. Fungus of thrush or Oïdium albicans, prepared in glycerin 213
69. Hypertrophy of the lingual tonsil 218
70. Pharynx viewed from behind (after *Toldt*) 234
71. The naso-pharyngeal space (after *Toldt*) (right lateral view) 235

FIG. PAGE
72. Hypopharyngoscopy (after *von Eicken*)................ 245
73. Suction apparatus for the tonsil...................... 251
74. Spongeholder for the throat and naso-pharynx.......... 253
75. Stricture of pharynx (after *Schech*).................... 257
76. Left-sided peritonsillar abscesses: Incision............... 268
77. Pharyngeal scissors with the blades bent on the flat (after *Cordes*).. 282
78. Tonsil squeeze (after *Hartmann*)...................... 283
79. Tonsil-splitter (after *M. Schmidt*)..................... 283
80. Adenoid vegetations.................................. 287
81. Adenotome (after *Beckmann*), in various sizes........... 290
82. Diagrammatic representation of adenotomy............. 291
83. Tonsillotome (after *Mathieu-Fahnenstock*)............... 295
84. Application of the tonsillotome........................ 296
85. Tuberculosis of soft palate............................ 309
86. Lupus of the soft palate (*Toldt*)....................... 310
87. Gummatous ulceration of the soft palate in the left tonsil and the posterior pharyngeal wall (*Türck*)........... 313
88. Typical naso-pharyngeal polypus (*Mikulicz*)............ 324
89. Ulcerating carcinoma of the left tonsil (*Türck*).......... 329
90. The larynx, viewed from behind, the muscles being removed... 343
91. The aditus laryngis, viewed from behind............... 344
92. Ligaments of the larynx. The left half of the larynx, viewed from within (*Toldt*)....................... 345
93. Frontal section of the larynx, viewed from behind (*Toldt*) 346
94. Sagittal section of larynx, left side (*Toldt*).............. 347
95. Muscles of larynx from behind (after *Störk*)............. 348
96. Muscles of larynx, lateral view (*Störk*).................. 349
97. The thyroid and arytenoid cartilages are cut horizontally.. 350
98. M. crico-thyroideus, viewed from in front and left side (*Toldt*)... 352
99. Nerves of the larynx (*Toldt*).......................... 354
100. Fusible cast of the bronchial tree, frontal view (after *Sahli*).. 357
101. Diagrammatic representation of the centers of respiration and phonation in the brain and medulla oblongata and their tracts (after *Réthi*).................... 362
102. Laryngeal mirror.................................... 364
103. Indirect laryngoscopy. Insertion of the mirror (frontal view).. 366
104. Laryngoscopia indirecta. Examination by mirror (lateral view).. 369
105. Omega-shaped epiglottis.............................. 370
106. Retroflexed epiglottis................................ 370
107. Holder of the epiglottis (after *Reichert*)................ 370
108. Relations of the laryngoscopical image to actuality...... 371
109. Vocal cords during phonation......................... 372
110. The same during respiration.......................... 372

FIG. PAGE
111. Same during deep inspiration........................ 373
112. Laryngoscopic image (enlarged)...................... 374
113. Arrangement for the examination by *Killian's* method... 375
114. Position of the mirror in *Killian's* method of examination 376
115. Posterior pharyngeal wall and bifurcation (*Killian*)...... 377
116. Subglottic laryngoscopy.............................. 377
117. Spatula of *Kirstein*................................ 378
118. Autoscopy (after *Kirstein*) (diagrammatic)............. 379
119. Intralaryngeal tubiform spatula (after *Killian*).......... 380
120. Universal demonstration electroscope (after *Caspar-Killian*)....................................... 380
121. Long sponge-holder for local anæsthesia................ 381
122. Tube for bronchoscopy............................... 382
123. *Killian's* syringe for aspiration of the mucus of the trachea or bronchi..................................... 382
124. Laryngeal probe.................................... 383
125. Laryngeal electrode.................................. 394
126. Sponge-holder for the larynx......................... 395
127. Painting the larynx.................................. 396
128. Laryngeal syringe................................... 397
129. Laryngeal insufflator................................ 397
130. Apparatus for inhalation............................. 398
131. Galvano-caustic burner for larynx..................... 401
132. Double needle for electrolysis......................... 401
133. Fibrous bands in the trachea (after *Türck*).............. 410
134. Annular stenosis of the trachea (after *Türck*)............ 410
135. Scabbard-shaped compression of trachea (*Hochenegg*) 411
136. Bilateral compression of trachea (image) (*Türck*)........ 412
137. Compression of the left side seen as image (*Schrötter*)...... 412
138. Tracheal compression by a retrosternal thyroid goitre.... 415
139. Tracheal dilator.................................... 418
140. Tracheal canula (*Lüer*)............................. 418
141. *König's* long tracheal canula......................... 420
142. Instruments for intubation (after *O'Dwyer*)............. 422
143. Intubation (after *O'Dwyer*) (schematic)................. 423
144. Extractor for *O'Dwyer's* tube......................... 424
145. Tube for catheterisation (after *Schrötter*)............... 427
146. Tin dilator (bolt) for obturation of the larynx and introducer (*Schrötter*)............................. 429
147. Canula fitted with *Schrötter's* tin bolt................. 430
148. Chimney-canula (after *Dupuis*) (one half natural size) ... 430
149. T-shaped canula.................................... 430
150. Subglottic laryngitis................................ 435
151. "Congestive" œdema of arytenoid cartilages and ary-epiglottic folds................................... 442
152. Laryngitis phlegmonosa circumscripta. Abscess of epiglottis (*Türck*).................................. 442
153. Unguarded laryngeal knives (after *B. Fränkel*).......... 445
154. Guarded laryngeal knife (after *Tobold*)................ 445

FIG. PAGE
155 *a*. Pachydermia laryngis........ 452
155 *b*. Thickening of the epithelium of the pad-like projection of the posterior wall........ 452
156. Chorditis nodosa (singer's nodes)........ 453
157. Chorditis hypertrophica superior........ 453
158. Double curette cutting vertically (after *Landgraf-Krause*) 457
159. Scooped forceps (after *Rosenberg*)........ 458
160. Right-sided perichondritis arytenoidea (*Türck*)........ 461
161. Left-sided perichondritis cricoidea (*Türck*)........ 461
162. Laryngeal diphtheria (*Türck*)........ 472
163. Broad-based infiltration of the posterior laryngeal wall... 483
164. Conical infiltration of posterior wall........ 483
165. Serrated infiltration and papillary excrescences of the posterior wall........ 484
166. Infiltration of the epiglottis, aryepiglottic folds, and posterior wall (*Türck*)........ 484
167. Tuberculous ulcers on both vocal cords and posterior wall 485
168. The left vocal cord appears divided into two parts by a notch-shaped ulcer (lip-ulcer)........ 485
169. Tumour-like ulcer of the right vocal cord, covered with granulations........ 486
170. The right vocal cord is irregularly infiltrated, giving the appearance of a tumour, with ulceration........ 486
171. Pressure forceps (after *Krause*)........ 493
172. Curette with revolving end for the posterior laryngeal wall. 494
173. Revolving double curettes (after *Krause-Heryng*) with guide tube and wire, fixed on a *Krause's* universal handle........ 495
174. Curettement of the right ventricular fold........ 496
175. Removal of the epiglottis........ 497
176. Lupus of the larynx (*Chiari*)........ 499
177. Condylomata of epiglottis (*Chiari*)........ 502
178. Gummatous infiltration of epiglottis (*Türck*)........ 503
179. Gummatous infiltration of right vocal cord (*Chiari*)...... 503
180. Gummatous ulcers on the vocal cord and aryepiglottic folds (*Türck*)........ 504
181. The same case as in Fig. 180 after healing up (*Türck*).... 504
182. Both vocal cords are notched or serrated by gummatous ulcers (*Schnitzler*)........ 505
183. Scleroma laryngis. Subglottic laryngitis; subglottic pads (*Türck*)........ 508
184. Subglottic knot of scleroma on the right vocal cord...... 508
185. Instruments for removal of foreign bodies (after *Killian*). 519
186. Fibroma of right vocal cord (*Türck*)........ 527
187. Same as in Fig. 186 during phonation (*Türck*)........ 527
188. Pedunculated fibroma of left vocal cord, arising from the upper surface........ 527
189. Same as in Fig. 188 during phonation........ 527
190. Papilloma of right vocal cord (*Oertel*)........ 528

FIG. PAGE
191. Multiple papillomata in larynx and on epiglottis (*Oertel*).. 528
192. Excessive development of papilloma in a child (*Oertel*)... 528
193. Cyst of epiglottis (*Mackenzie*)........................ 529
194. Cyst arising from the right ventricle (*Bruns*)............ 529
195. Revolving scooped forceps, adaptable to *Krause's* universal handle.. 535
196. Sessile sarcoma of the left ventricular fold.............. 539
197. Pedunculated sarcoma of the right vocal cord (*Türck*) ... 539
198. Small tuberous carcinoma of right vocal cord (*Jurasz*) ... 543
199. Carcinoma covering the entire left vocal cord and showing ulceration (*Türck*).............................. 543
200. Carcinoma of the left side of larynx, showing ulceration at the posterior wall, spreading on to the epiglottis (*Türck*).. 544
201. Carcinoma of the left side of larynx, showing ulceration, perichondritis, and oedema (*Türck*)................ 544
202. Carcinoma of right half of larynx, spreading to the pharynx and tongue (*Türck*)...................... 546
203. Carcinoma of lower pharynx (*Türck*).................. 546
204. Hysterical paralysis of the adductors on phonation 560
205. Hysterical paralysis of adductors. Vicarious adduction of ventricular folds (*Chiari*)...................... 560
206. Paralysis of left m. thyro-arytenoideus internus during phonation.. 566
207. Bilateral paralysis of mm. thyro-arytenoidei interni during phonation.. 566
208. Paralysis of m. arytenoideus transversus during phonation. 566
209. Paralysis of the m. arytenoideus transversus and both mm. thyro-arytenoidei interni during phonation.......... 566
210. Paralysis of left crico-arytenoideus posticus during respiration (*Réthi*)................................ 568
211. Paralysis of both the crico-arytenoidei postici. Respiration 569
212. Paralysis of both the crico-arytenoidei postici. Phonation 569
213. Paralysis of left recurrent nerve (during respiration)..... 571
214. Paralysis of left recurrent nerve (during phonation)...... 571
215. Paralysis of left recurrent nerve (during phonation)...... 572
216. Bilateral paralysis of the recurrent nerve symmetrically on both sides.................................... 572
217. Points of stimulation of the laryngeal muscles (after *Ziemssen*).. 576

PART I.

Diseases of the Nose and Its Accessory Cavities.

PART I.

Diseases of the Nose and Its Accessory Cavities.

GENERAL SECTION.

ANATOMY.

THE EXTERNAL NOSE.

The shape of the nose is outlined by a skeleton composed of osseous and cartilaginous elements.

To the former belong the *nasal processes of the superior maxillae,* which ascend like wings, and the so-called *nasal bones* proper, which are connected with the nasal process of the frontal bone and thus form the root and the bridge of the nose.

To these bones are firmly attached a few hyaline cartilages.

The *triangular cartilage* passes from the anterior margin of the *cartilaginous septum* laterally on both sides.

To these are attached the *wing cartilages,* which turn round the nostril like a horseshoe. Mesially, between the two wing cartilages, can be felt, in a shallow groove, the lower end of the *cartilaginous septum.*

Between the triangular and wing-shaped cartilages a few sesamoid cartilages are interspersed on either side.

The lower margin of the triangular cartilage turns a little towards the inner side, forming a ridge, called the plica vestibuli (vestibular fold), which separates the vestibulum nasi from the nasal cavity proper.

This walled-in space is sometimes called the inner nostril.

Upon the cartilages lie the muscles which dilate or constrict the nares—levator and depressor alae nasi.

The outer nose is very vascular. The arteries are derived mostly from the facial artery. *The veins are connected with those of the nasal mucous membrane* and enter the facial vein.

The nerves are branches of the *seventh* (facial), which supplies the muscles; the infraorbital branch of the second division of the fifth, which is the sensory nerve of the bridge and alae nasi; and also the nasal branch of the first (ophthalmic) division of the fifth, which is the sensory nerve of the point of the nose.

The skin of the outer nose, especially at the tip and the wing, contains numerous sebaceous glands, which often show retention of their secretions, and then are known as comedones. The skin is reflected inwards to line the introitus of the nose up as far as the plica vestibuli and the *anterior end of the lower turbinal.* Especially in elderly persons, particularly men, stiff hairs, known as *vibrissae,* are found in the introitus, which not infrequently may give rise to furunculous inflammation.

THE INTERNAL NOSE.

Nasal Cavity.—The nasal cavity is divided by a septum into two halves, which open through the two *choanae* into the nasopharynx.

The dividing partition (*septum nasi*) consists of two bony parts: the one is the vomer, which extends from behind forwards from its broad base at the choanae to its point in front; above and in front of it is the other, the perpendicular lamina of the ethmoid.

Attached to the osseous elements is the cartilaginous portion of the septum—the *quadrangular cartilage*—which forms the most anterior part of the septum and often presents deviations. (See Fig. 1.)

The anterior border of the quadrangular cartilage is always covered by skin.

At a point on the septum, corresponding to the anterior end of the middle turbinal, is a part of the mucous membrane which is often thickened by a conglomeration of sebaceous glands, called the *tuberculum septi.*

An organ on the septum of great biological interest, but in man only rudimentally developed, is that known as the organ of *Jacobson,* called the "*corpus vomero nasale*" (or vomero-nasal body), which is situated in front and below the tuberculum and forms a tubular structure containing nerve filaments.

The septum is at right angles to the roof of the nose, at the *lamina cribrosa,* which is the thinnest part of the basis cranii.

Behind the lamina cribrosa, and forming an angle with it, the roof of the nose is formed by the anterior wall of the sphenoidal sinus. (See below.)

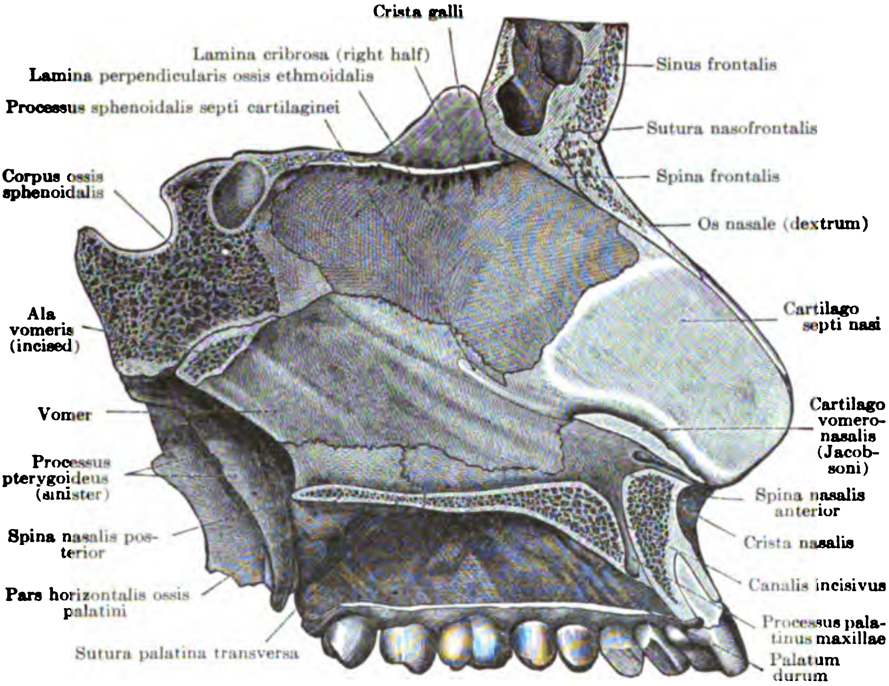
Crista galli
Lamina cribrosa (right half)
Lamina perpendicularis ossis ethmoidalis
Processus sphenoidalis septi cartilaginei
Corpus ossis sphenoidalis
Ala vomeris (incised)
Vomer
Processus pterygoideus (sinister)
Spina nasalis posterior
Pars horizontalis ossis palatini
Sutura palatina transversa
Sinus frontalis
Sutura nasofrontalis
Spina frontalis
Os nasale (dextrum)
Cartilago septi nasi
Cartilago vomeronasalis (Jacobsoni)
Spina nasalis anterior
Crista nasalis
Canalis incisivus
Processus palatinus maxillae
Palatum durum

Fig. 1.—The osseous and cartilaginous skeleton of the septum nasi (viewed from the right side) (*after Toldt*).

Of great importance is the anatomy of the lateral nasal wall.

The underlying structure of the lateral nasal wall is the inner surface of the superior maxilla.

The large opening in the superior maxilla—*hiatus maxillaris*—is narrowed through the attachment of various bones in such a way that only a small hole remains, the *ostium maxillare,* through which the maxillary cavity—the antrum of Highmore—communicates with the nasal cavity.

The narrowing of the hiatus is partly due to the palate bone. Chiefly through the processus ethmoidalis of the inferior turbinal uniting with the processus uncinatus of the ethmoidal bone, which descends from above and in front, downwards and backwards. (See Fig. 2.)

Essential in regard to the topography of the nasal cavity are the three turbinals; the *upper* and *middle* of which belong to the ethmoidal bone, the *lower* being a structure of itself. The *superior turbinal* (concha superior) is so hidden that it cannot be seen by anterior rhinoscopy. Its anterior end is continuous with the middle turbinal, which lies immediately below.

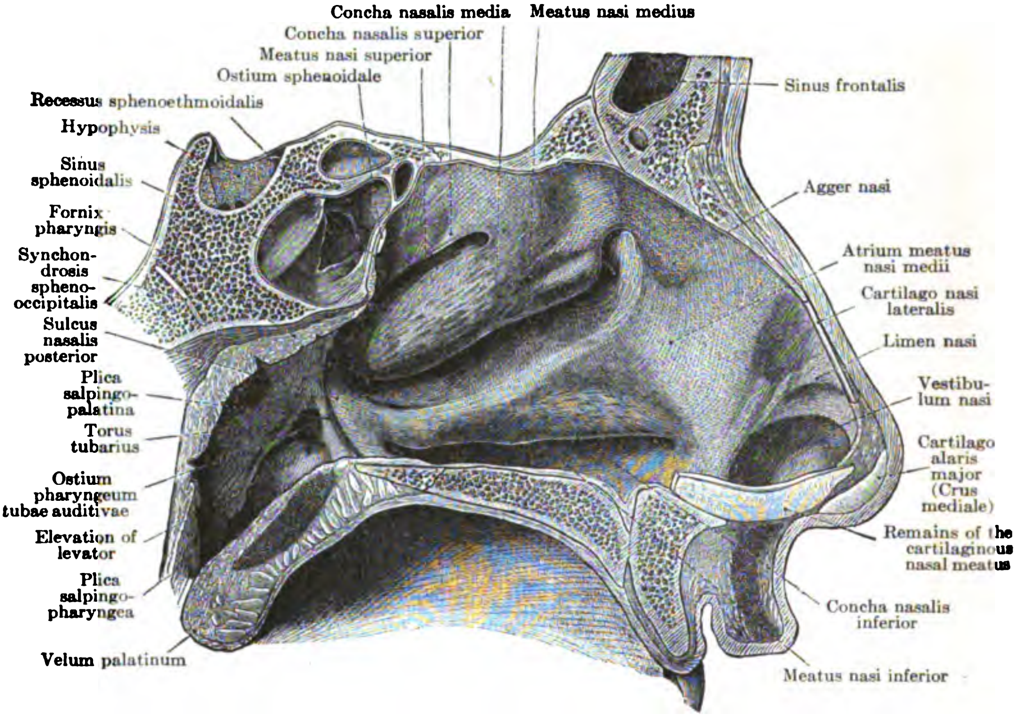

Fig. 2.—The left lateral wall of the nasal cavity, showing the turbinals and nasal meatus (*after Toldt*).

The *middle turbinal* (concha media) has a length of 20 mm. or more and projects free into the cavity of the nose by its anterior end, or operculum, 10 to 12 mm.

The small space between the middle turbinal and the septum is called the olfactory chink (rima olfactoria). The *inferior turbinal* (concha inferior) is attached by its maxillary process to the nasal surface of the superior maxilla, and its lower free margin protrudes into the nasal cavity. Its size varies much. The length is between 25 and 50 mm.

Between the upper and middle turbinals is the *superior meatus;* between the middle and lower turbinals is the *middle meatus*, and between the lower turbinal and the floor of the nose is the *inferior meatus* of the nose.

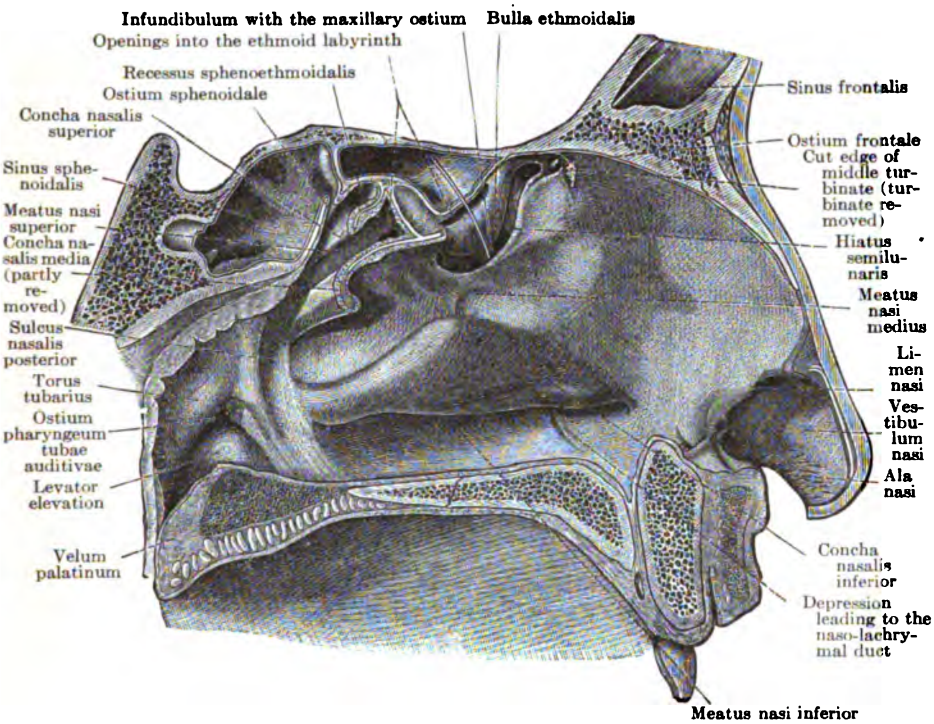

Fig. 3.—Left lateral wall of nasal cavity. The greater part of the middle and the anterior end of the upper turbinals are removed (*after Toldt*).

Into the inferior meatus, about 10 mm. behind the anterior end of the inferior turbinal and immediately below its attachment, is the opening of the *naso-lachrymal ducts* (ductus nasolachrymalis) (Fig. 3).

In the middle meatus are several spaces, varying in size, number, and form, and covered with mucous membrane, the most important of which is the *hiatus semilunaris*, running curvilinearly from above and in front downwards and backwards; this expands downwards in a funnel-shaped manner, forming the *infundibulum*. Into the infundibulum opens above, the frontal sinus by the *ostium frontale;* and a little below and behind opens the antrum of *Highmore* by the ostium maxillare; between these two are the several openings of the anterior ethmoidal cells.

The *bulla ethmoidalis*, a boss-like expansion of the ethmoidal bone, overhangs the hiatus semilunaris, varying very considerably in size, so that the anatomical configuration of this region is very much influenced by it.

Besides the *frontal* and *maxillary* cavities and the *anterior ethmoidal cells*, the *posterior ethmoidal cells*, and *sphenoidal cavity* are in communication with the nasal fossae.

Accessory Nasal Cavities.—The *sinus maxillaris* (antrum Highmori) has the shape of a pyramid, the base of which corresponds to the outer wall of the nose, and the apex towards the malar bone. The surfaces of the pyramid are formed by the floor of the orbit, the anterior or facial, and the posterior or lateral wall of the superior maxilla. The anterior wall below the infraorbital foramen is more or less depressed, forming the canine fossa (Fig. 4).

Where the facial and nasal walls of the antrum join, a broad groove is formed, running from before backwards, which lies in some cases considerably deeper than the floor of the nose, and *Zuckerkandl* describes such a deep excavation of the alveolar process as the *sinus alveolaris*. This depression represents the most usual expansion of the antrum.

On the other hand, the floor of the maxillary cavity might be on a level with, or higher than that of, the nasal cavity. In any case the antrum varies in size and shape very frequently, and there are cases where both cavities differ in size. Generally, the thinner its walls, the larger is the antrum.

The sinus alveolaris might be so excessively developed that the roots of the teeth project into the maxillary cavity.

The osseous alveoli in such a case might be so thin that on extraction of a tooth the antrum might be opened.

In some unique cases the alveoli show small canals for vessels and nerves, or larger defects through which the root of a tooth might be free in the antrum.

The cavity is sometimes divided into two parts by a vertical, more rarely by a horizontal, partition.

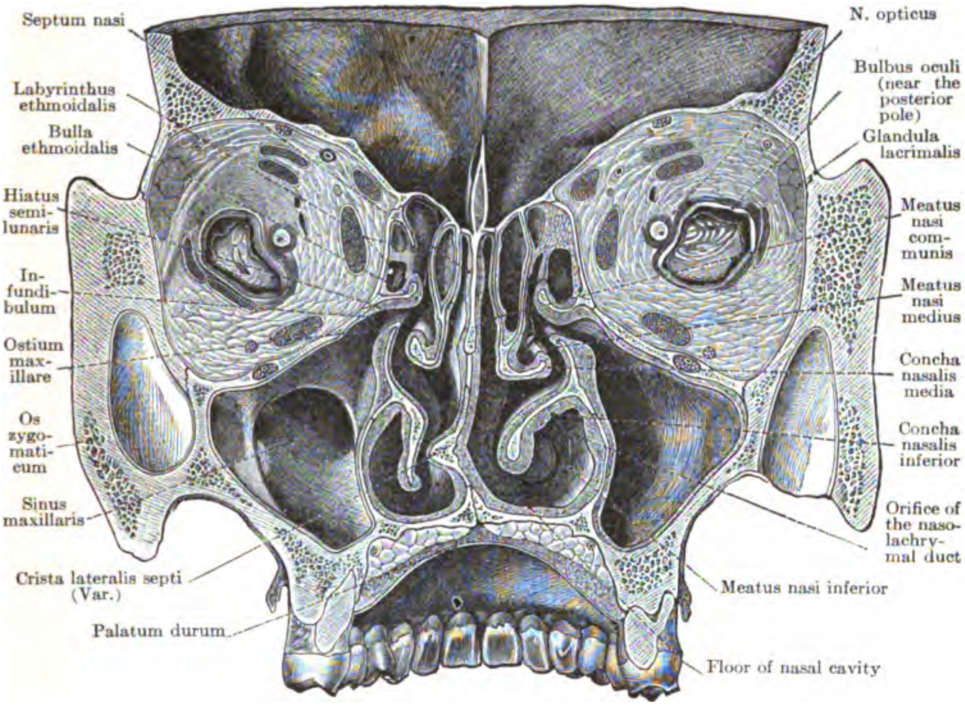

Fig. 4.—Frontal view of the nasal cavity. The coronal section is through the middle of the nose, showing the anterior section from behind. Opening of the maxillary cavities. The orbits are cut through immediately behind the entrance of the optic nerve into the sclera (*after Toldt*).

The maxillary opening (ostium maxillare), the position of which has already been described on page 6, viewed as from within, lies immediately below the floor of the orbit and varies as regards shape and size. Sometimes there is a second opening, called the *foramen accessorium,* in the middle or inferior meatus.

The *frontal sinus* (sinus frontalis) has the form of a three-sided pyramid, the apex of which is directed towards the forehead; the base is formed by the junction of the squamous and orbital parts of the frontal bone.

It lies over the root of the nose, in the lower part of the squamous portion of the frontal bone, and extends variably in all directions. The cavity in adults extends from the middle line as much as 5 cm. Sometimes the lumen is markedly small, and even entirely absent.

The frontal sinus is divided in the median line by a more or less thick, sometimes perforated, septum, into two halves.

The partition, however, is not always in the middle line, so that the two halves are often quite asymmetrical.

The cavity opens below by means of the naso-frontal duct (ductus naso-frontalis), behind the anterior end of the middle turbinal bone.

The mouth of the duct, the ostium frontale properly so called, lies in the hiatus semilunaris, above and in front of the ostium maxillare.

The *ethmoidal cells* (cellulae ethmoidales) form a labyrinth of thin-walled intercommunicating cavities, varying in shape, size, and number, which are separated from the orbit by the lamina papyracea (os planum).

One differentiates anterior and posterior ethmoidal cells. The *anterior* cells open by several apertures (foramina ethmoidalis) into the hiatus semilunaris of the middle meatus, behind the ostium frontale. The *posterior* cells open into the superior meatus, or are in direct communication with the sphenoidal sinus. By this way one can reach the sphenoidal sinus directly from the ethmoidal labyrinth. Sometimes one or the other of the anterior cells bulges towards the frontal sinus and its duct or lies in front of the latter; which relation might be of importance when using the probe.

Of special importance is the bulla ethmoidalis, already mentioned on page 6.

It is actually an ethmoidal cell which convexedly protrudes into the middle meatus laterally from the lamina papyracea. It usually contains a cavity, sometimes of quite large dimensions, so that the turbinal bone might be pushed towards the septum nasi, causing a deviation of the latter.

For the relation of the bulla ethmoidalis to the hiatus semilunaris, see page 6.

The *sphenoidal sinus* (sinus sphenoidalis) lies in the upper and back part of the nasal cavity, above the choanae, within the body of the sphenoidal bone. It varies in its dimensions still more than the other accessory cavities; sometimes it might be abnormally small, another time excessively large, and in such a case it might extend into neighbouring bones. Sometimes the sinus is altogether missing, and only a slight recess on the anterior wall of the sphenoidal body, which is lined by nasal mucous membrane, is all that in other conditions represents the sphenoidal sinus.

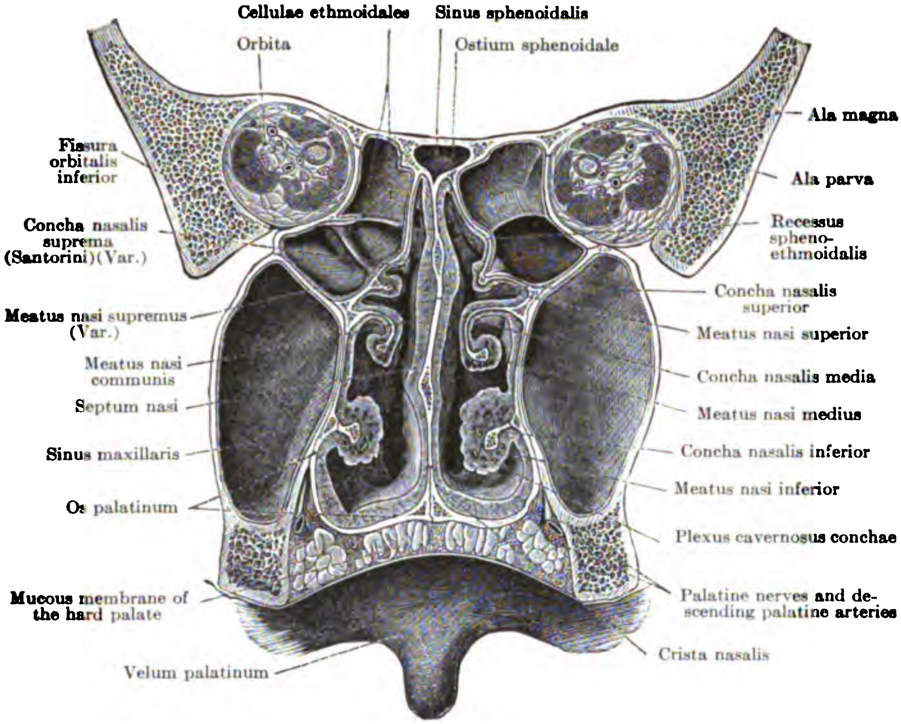

Fig. 5.—Frontal section through the posterior part of the nasal cavity and maxillary sinus. The posterior section is viewed from in front. Opening of the sphenoidal cavities (*after Toldt*).

The sphenoidal sinus is divided into two or more unequal parts by septa, or there is only one cavity, owing to absence of any septum.

The *anterior* walls of the sphenoidal sinus, which looks towards the nasal cavity, and the *upper* wall, which carries the *optic chiasm* and the *hypophysis cerebri*, are relatively thin.

This explains the liability of the eye and the orbit to become involved in inflammatory processes of the sphenoidal cells. The lateral walls are usually very thin; thicker is the lower wall, which forms the roof of the hindermost part of the nasal cavity and naso-pharyngeal space.

The anterior wall of the sinus might be partially or totally absent, and in this case the ethmoidal cells are continuous with it.

Embryologically the sphenoidal sinus must be considered as really the hindermost of the posterior ethmoidal cells.

At the point where the anterior wall of the sphenoidal cavity meets laterally with the posterior end of the ethmoidal bone, is found a depression of the nasal mucous membrane called the *recessus spheno-ethmoidalis;* into this recess opens the sphenoidal sinus, by the *ostium sphenoidale.*

If investigated from within the nasal cavity, the opening is found as a minute aperture immediately below the roof of the nose, somewhat above the posterior end of the middle turbinal, and a little laterally.

The Mucous Membrane of the Nose.—The nasal mucous membrane can be differentiated into three sharply distinguishable sections, which are also different in regard to their function.

The first section, which lines the vestibulum nasi, is merely the continuation of the external skin, reflected round the cartilages into the introitus, and is covered for this reason by several layers of *squamous* epithelium. (See Fig. 6, septum nasi.)

The following section, covering the greatest portion of the nasal and the accessory cavities, shows the characters of the respiratory mucous membrane. It is lined with *ciliated* epithelium and characterized by its light red color.

The third and smallest portion, carrying in its substance the specific terminations of the olfactory nerve, is more yellow or yellowish-brown; its epithelium is thicker than that of the respiratory portion, and is composed of *olfactory cells, supporting cells,* and *basal cells.*

It is interesting to note that just this portion of the nasal mucous membrane which forms the morphological basis of the sensory organ peculiar to the nose, is limited to so small a region. This can be taken as a proof of the little delectable fact that the sense of smell in Homo sapiens is on a retrograde movement, or seems to degenerate entirely, in contradistinction to his animal affinities.

In the *olfactory region* (regio olfactoria) some tubular glands (glands of *Bowman,* glandulae olfactoriae), and in the *respiratory region* (regio respiratoria) acinous glands, in enormous numbers, are found.

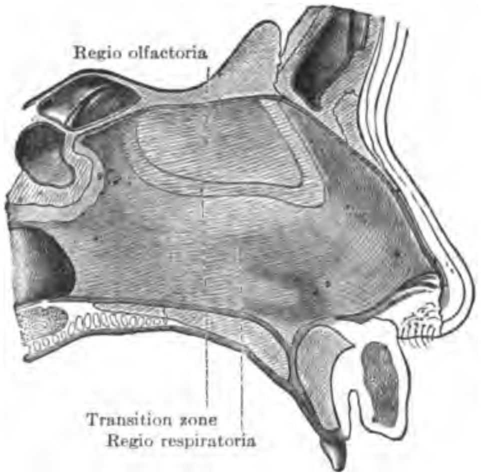

Fig. 6.—Septum of the nose.

Blood-vessels.—The nasal mucous membrane is very vascular. The arterial blood-supply is derived chiefly from the spheno-palatine artery (arteria spheno-palatina), which, arising from the internal maxillary artery, itself gives off the *posterior* and *lateral* nasal arteries to the lateral wall of the nasal cavity, and the *posterior* nasal arteries to the septum. The septum receives also branches from the facial artery.

Lastly, the ophthalmic artery from the internal carotid sends the *anterior* and *posterior* ethmoidal arteries to the mucous membrane.

The *veins* accompany the arteries and open into the *facial* and the *ophthalmic veins;* but communicate also, through the *lamina cribrosa*, with the *veins of the dura mater and longitudinal sinus.*

The veins form a dense network in the mucous membrane, especially on the turbinal bodies, where the tissue assumes a cavernous character, due to a peculiar formation of the veins themselves, and sinus-like dilatations of their lumina; in that respect they are spoken of as *cavernous bodies.* The cavernous bodies, therefore, as can be seen, are interposed into the venous system.

The fibrous tissue, wherein the spongy cavernous tissue is embedded, is rich in *elastic* and *muscular* fibers; which explains the rapid swelling, on and off, of the mucous membrane, a process which is governed by the trigeminal nerve, especially by the fibers coming from the spheno-palatine or *Meckel's* ganglion. The cavernous bodies are, according to *Zuckerkandl*, found in greater bulk where the mucous membrane comes in contact with a large quantity of air; that is, on the entire lower turbinal body, on the lower margin of the middle turbinal, and on the posterior ends of the middle and superior turbinals.

Some authors describe also cavernous tissue as being found on the septum and tuberculum septi. Probably this is only an excess of glandular tissue in that position.

The cavernous bodies have the function of warming the inspired air, perhaps also of keeping the mucous membrane humid.

Lymph-vessels.—The lymph-vessels of the nasal mucous membrane form an extremely dense network, which, according to *Axel Key* and *Retzius*, communicate with the *subdural* and *subarachnoidal space;* but this is denied by *Zuckerkandl.*

Nerves.—The nerves of the nasal mucous membrane are derived from the *olfactory* and *trigeminal.* The first is limited exclusively to the olfactory region; the latter supplies the olfactory chink (rima olfactoria) and the entire respiratory region.

The *olfactory bulb* (bulbus olfactorius) lying on the lamina cribrosa sends very fine filaments through the holes of the cribrous plate, which ramify and descend along the septum and inner surfaces of the superior turbinal, and terminate in short processes, called *smelling-hairs*, within the olfactory cells.

The fifth nerve provides, from its first and second division, the sensory nerves for the nasal mucous membrane. The *secretory* fibers are derived, according to *Aschenbrandt*, from the spheno-palatine nerve of the second division.

PHYSIOLOGY.

The nose is not only an organ of smell, but plays an important rôle in respiration, as an air conduit. Both functions are in a certain connection with one another. The perception of smell depends on the possibility of conveying stimuli to the olfactory chink by means of the air current. If breathing be stopped, no smell can be perceived, however volatile the odorous substance may be.

Under normal conditions the air current in respiration passes through the nose exclusively. Mouth-breathing is any way pathological. The direction taken by the air current in inspiration—as *Burchardt* has recently shown on a bisected skull, artificially fitted with soft parts, and corroborated by *Réthi*—goes from the nostril upwards through the middle meatus; and small deflections and irregularities promote the conveyance of odourous substances to the olfactory region. On its way through the nose the air is warmed, moistened, and cleaned. According to *Aschenbrandt* and *Kayser*, the air is warmed up to 30° C., from moderate outdoor temperature; and is completely saturated with moisture. Both warming and moistening of the air are done by the cavernous bodies of the mucous membrane (see page 12). The air is not completely cleaned by the nose, but anyhow a great deal of the dust and other impurities are precipitated upon the moist mucous membrane, whereas the remainder is deposited on the posterior pharyngeal wall, opposite the choanae. Along with the dust, bacteria also are retained; hence the normal mucus is poor in microbes. Whether the nasal mucus, as is contended, has a bactericidal action is doubtful, and is certainly not proved by the fact that the nose forms the door of entrance for so many pathogenic organisms. It is certain that the protection afforded by the nose is not absolute. According to *Schousboe*, bacteria are much more numerous in the vestibule than within the nasal cavity.

Smelling is a very complicated function, and various points must be considered. We smell not only during inspiration, but also during expiration. The admission of odorous particles through the choanae is, according to *Nagel*, more important than smelling through the nostrils. 'Expiratory smelling is most pronounced during the act of swallowing; thereby volatile particles invade the nose from behind and excite sensations which are frequently mistaken for gustatory perceptions. *Zwaardemaker* speaks of a "gustatory smelling" in this sense.

Whether the trigeminal nerve takes part in smelling is not yet decided; but is probable from the investigations by *Magendie* and *Krause*. This nerve probably has the task of bringing to perception sharp, irritating, or acrid gaseous particles.

In total anosmia acrid substances, such as sal ammoniac, vinegar, formaldehyde, etc., are still perceived, perhaps as smell; although the sensory fibers of the trigeminal nerve may conduct the stimuli.

In order to excite the sensation of smell, odorous substances must be gaseous or in such a physical state that they can mix themselves actively or passively with the current of air (*Gaule*). By prolonged excitation the sense can be so influenced that it becomes fatigued, and only after several minutes does the olfactory nerve once more recover functionally. The simultaneous influence of several odours produces a mixed sensation of smell, or a single odour might abolish the others. In children the sense of smell is very acute, but in old age it is diminished, and decreases more and more.

Now a few words about the nose as a *reflex-exciting organ.* The reflex irritability of the nasal mucous membrane is very marked, owing to the abundance of its sensory nerves. From every part of the nasal mucous membrane a reflex can be excited; and, according to *Sandmann*, most easily from the so-called "irritable zones." These lie on the anterior and posterior ends of the middle and lower conchae (turbinals) and the corresponding sites on the septum. Best known is the *sneezing reflex*, which acts, so to speak, as the watch-dog of the respiratory tract. If the sensory fibers of the nasal nerve, a branch of the ophthalmic division of the trigeminal, are stimulated by a foreign body or mechanically by chemicals or by pathological processes in the mucous membrane, the stimulus is centripetally conducted to the medulla, and thence centrifugally to the motor nerves of the soft palate and the expiratory muscles, and then produces a sudden explosive expiration which forces open the closure of the throat and nasal cavity and drives the air current forcibly through the nose solely. Recent investigations which *Nagel* and other authors have made tend to show that the expiration passes not through the nose, but through the mouth. Simultaneously the secretions of the mucous membrane are stimulated, and the shrinking caused thereby produces a subjective feeling of freeness within the nasal cavity as the result of the sneeze itself, and not solely on account of the ejection of the secreted mucus. Just as in sneezing the motor nerves are brought into action, so in lachrymation secretory nerves, and in the swelling and shrinking of the cavernous tissue vasomotor nerves, are concerned. The irritability of the nasal mucous membrane is also the physiological basis of a whole series of pathological reflexes which will be discussed later in the chapter on Reflex Neuroses.

The nose plays an important rôle as a *voice-* and *speech-producing organ*, in so far as, together with the naso-pharyngeal space, it forms the resonator for the sounds produced by the larynx. The vibrations of the air-column in the nose and naso-pharyngeal space increase the sound and accord the characteristic timbre; hence obstruction of the naso-pharyngeal channel tends to alter the sound of the voice.

The function of the accessory cavities is, as the matter stands, not yet clear. According to one theory, they are supposed to take part in the act of smelling; and according to another theory, they contribute to the warming of the inspired air. The extraordinary variability of the accessory cavities in man proves that they are not very important from a physiological point of view.

METHODS OF EXAMINATION.

Examination of the *external nose* consists of inspection and palpation.

Examination of the *nasal cavity* includes the following: (1) Examination from in front. (2) Examination from behind: *e. g.*, from the choanae, combined with examination of the naso-pharyngeal space. (3) Transillumination of the various accessory cavities of the nose.

THE EXAMINATION FROM IN FRONT.

This consists of testing the function (permeability and sense of smell), observing any special odour (foetor); inspection (rhinoscopia anterior), as well as probing; and eventually resorting to local anaemia and local anaesthesia; in order to reduce the volume and sensitiveness of the mucous membrane.

The permeability of the nose with respect to air is tested by shutting off one nostril and then estimating the strength of the air current escaping through the other nostril by means of the hand held in front; or, by placing in front of the nostril a cold mirror and observing the condensation of the exhalation thereon (*Zwaardemaker*).

On the obstructed or narrowed side the condensation area is smaller. One must listen at the same time for a hissing noise which may be heard in congenital narrowing of the nose, and to the patient's voice with regard to quality of speech, which is often altered in these cases.

The test of the sense of smell—with regard to quality—is carried out by resort to various scents placed within each nostril separately (carbolic acid, iodoform, perfumes, etc.), and with regard to quantity—so far as the practitioner is concerned—is carried out by means of *Zwaardemaker's* olfactometer.

Foetor from the nose of patients is perceived by the examiner's own sense of smell, dealing with each nostril separately.

For anterior rhinoscopy a good light is necessary, and this may be either an oil lamp, incandescent, gas, or electric light. A concave mirror reflector, centrally perforated, serves the purpose of concentrating the light. It should have a focal distance of about 15 to 20 cm. (6 to 8 inches) and a diameter of 9 to 12 cm. (4 to 5 inches). The reflector is fixed by means of a band or ring to the forehead, so that the central hole comes just opposite the observer's (better) eye. Both physician and patient should be seated, the latter in such a way that the light is on the same level as the ear and on the same side as the reflector. If one uses a reflector fitted with an electric lamp in the center, there is more ease and freedom of action.

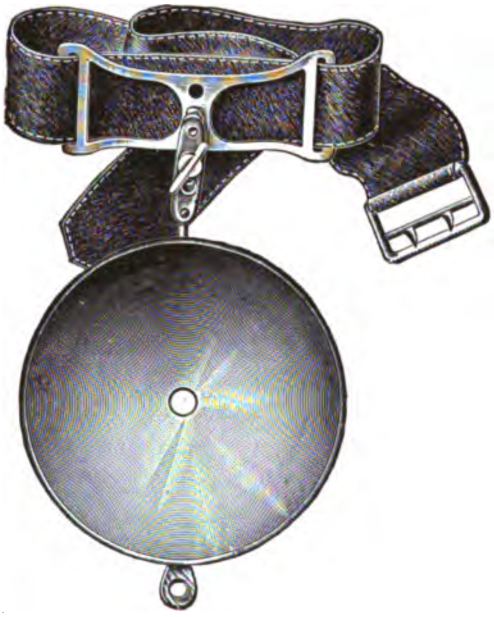

Fig. 7.—Reflector (*after Fränkel*).

Besides an incandescent gas flame within a porcelain shade, an electric head-lamp which is fitted with a small perforated reflector (after *Kirstein*) will be found useful.

By manipulation of the reflector the light is thrown into the nostril; the head of the patient is then slightly bent backward and the point of the nose tilted upwards with the finger in order to inspect the entrance of the nose.

If need be, it can also be inspected by a laryngeal mirror held in front and manoeuvered as is necessary. This done, the nasal speculum may be introduced in order to dilate the entrance. Various specula are in use, the most common being those of *Duplay, Kramer-Hartmann, Fränkel, Jurasz.* I prefer *Kramer-Hartmann's* speculum—for children, a correspondingly smaller size—the blades of which are easily opened. With children and infants one has very often to omit the insertion of a speculum.

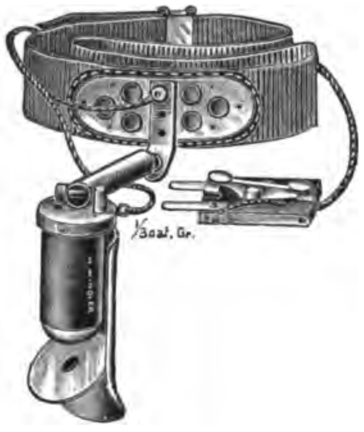

Fig. 8.—Electric head-lamp (*after Kirstein*).

The speculum is seized with the left hand, so that the handles are directed downwards, and the back of the hand is turned towards the mouth of the patient; then the closed instrument is inserted, about 1 to 1½ cm. into the nose, the point of which is pushed upwards; and the two blades are separated by adequate pressure of the hand, so that the patient does not experience discomfort. Special care should be taken at the insertion and opening of the speculum if the entrance of the nose be fissured or covered with scabs. The right hand of the examiner is placed on the forehead or occiput of the patient. The patient's head is then inclined forwards so that one can inspect through the speculum the floor of the nose, the lower meatus and concha, and also the lowest part of the septum. In this position, which is called the *first position*, the speculum lies nearly horizontally. In order to inspect the foremost part of the septum the speculum is turned outwards for ninety degrees, so that the two blades are not opened from right to left, but upwards and downwards.

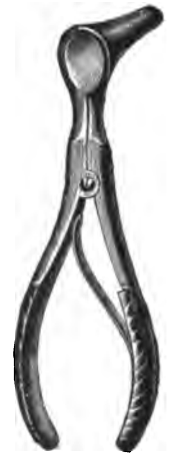

Fig. 9.—Nasal speculum (*after Kramer-Hartmann*).

This method of introduction is also useful in patients who suffer from nose-bleeding, because the bleeding point is nearly always seated on the foremost point of the septum, and could easily be injured by the pressure of the point of the inner blade. (See Fig. 10.)

If the nostril is wide enough, one is able to see in the depths a small part of the posterior pharyngeal wall, and on intonation of *i* or *u*, the movements also of the pad of the levator palati. (See Part III, p. 235.)

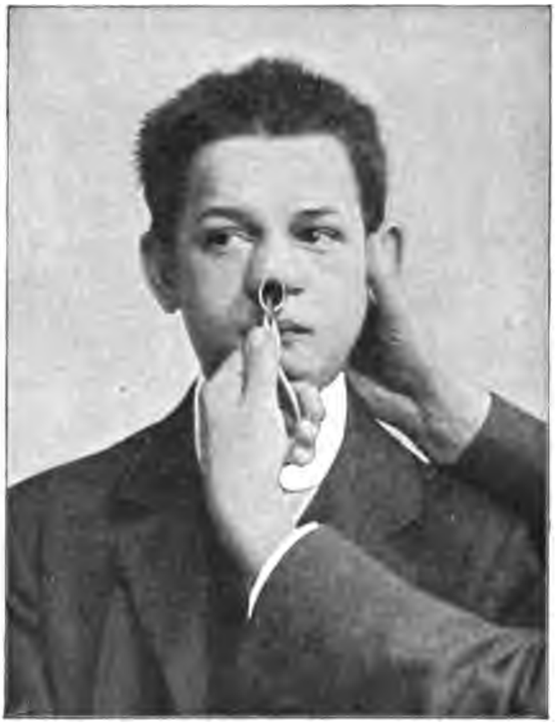

Fig. 10.—Anterior rhinoscopy. First position. The speculum lies horizontally.

The head of the patient is then reclined backwards and the speculum turned upwards; which movement brings into view the middle meatus and concha and the upper part of the septum, and sometimes also the roof of the nose. This is called the *second position*. (See Fig. 11.)

In order to have an easy survey of the topographic conditions of the middle meatus, *Killian* has constructed a speculum with very long and narrow blades, to be introduced after previous cocainization between the middle turbinal and the septum. This method of *rhinoscopia media* (middle rhinoscopy) (see Fig. 12) is very useful in the diagnosis of diseases of the accessory cavities.

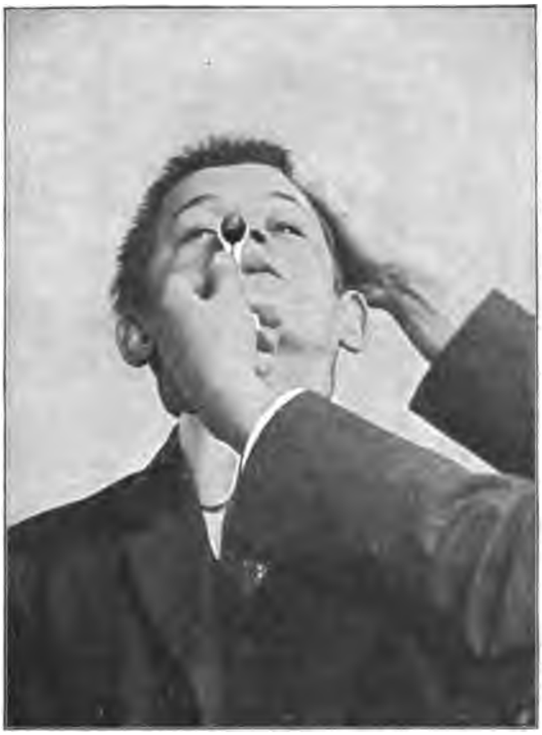

Fig. 11.—Anterior rhinoscopy. Second position. The speculum is turned upwards and a little obliquely.

For the beginner it is at first a little difficult to orientate, because the parts in the interior of the nose are all more or less foreshortened, and in the majority of cases show deviations from the normal, which even prevent illumination. The best guiding points are the anterior ends of the lower and middle concha. If the patient turns the head, we are sometimes enabled to see well into the depths along and between the lower concha and the septum.

Straight and wide noses admit an unimpeded inspection, and, as the case may be, a view of the upper margin of the choanae, the tubar pad, and even the ostium of the tube.

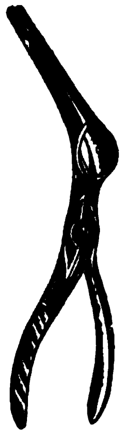

Fig. 12.—Speculum for middle rhinoscopy (*after Killian*).

Probing (Examination by the Probe; Sounding).—A probe of flexible metal, copper, or silver is indispensable for examination of the interior of the nose. The head of the probe must be blunt and smooth, so as not to hurt or irritate the very sensitive mucous membrane. The probe facilitates inspection of the nose, by pushing aside lengthy vibrissae which may obstruct the entrance of the nose or bulging of the mucous membrane or movable swellings of the conchae—of course, under guidance of the eye. It often happens that on mere touch with the probe the cavernous tissue of the conchae shrinks so much that the interior of the nose can be seen to greater extent than before. Accumulated secretions can also be loosened and may then be more easily blown out.

Douching in order to remove secretions should be used only in cases of need.

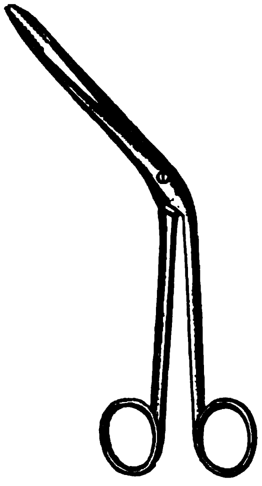

Fig. 13.—Nasal forceps (*after Hartmann*).

Anaesthesia and Artificial Anaemia (Anaemization).—In order to make inspection easier it is advisable to cause the mucous membrane to shrink by painting it with a solution of adrenalin 1 : 1000. It is sufficient to paint the mucous membrane several times with a swab on a holder, soaked with the solution; or, if the patient be nervous, to insert a pad of cotton-wool soaked with the solution for one or two minutes; which can be done by means of the nasal forceps or elbowed forceps. (See Fig. 13.)

Provided that the adrenalin solution be fresh, the mucous membrane will be rendered completely anaemic by it. It looks quite white and shrunken. If we now desire to render the mucous membrane insensitive also, we paint again with a little of a 10 to 20 per cent. cocaine solution by means of a swab. It is very convenient to mix 4 c.c. of a 1 : 1000 adrenalin solution with 36 c.c. of a 10 per cent. solution of cocaine for the purpose of painting the mucous membrane.

The previous or simultaneous application of adrenalin reduces very much the danger of cocaine poisoning, which was formerly observed frequently if cocaine was used solely.

If one inserts a cotton-wool swab with cocaine solution, one should take care that the patient always inclines the head forward, so that none of the solution shall run down into the pharynx. In children and patients suffering from heart disease it should be very cautiously applied. Symptoms of poisoning are pallor, discomfort, palpitation, sensation of cold, sweating; more serious symptoms are: oppression, nausea, vomiting, fainting. All the symptoms may after a time disappear of themselves. The treatment of the poisoning is merely symptomatic—the recumbent position, fresh air, smelling salts, brandy, and warm applications. Amyl nitrite has shown itself useful. (℞. Amylii nitrosi, Spir. aetheris nitrosi, āā 5.0. F. M. Several drops on a pocket-handkerchief for inhalation.)

Substitutes for cocaine are: Beta-eucaine, eucaine lactate, anaesthesin, yohimbin, stovaine, novocaine, and alypin. I possess real experience of only the last (alypin), and use it in 10 per cent. solution, which is equal to the more poisonous cocaine without causing the mucous membrane to shrink—an effect which is sometimes desirable in certain operations. The only disadvantage I have seen from the use of it is an increased watery secretion. As for cocaine, so for adrenalin also, other substitutes are in use, all of which contain the active principle of the adrenal glands.

I use for certain purposes *renoform*, which is the powdered dry extract of the adrenal gland, suspended in a solution of boric acid and milk-sugar. This mixed borated renoform solution has the advantage of infinite durability, in opposition to adrenalin solution, which is easily decomposed by air and light.

The accessory cavities can be sounded only after previous anaesthetization and anaemization; and should be done without using any force. The probe should be bent according to the purpose for which it is required. In atrophic rhinitis sounding is easy, whereas it is rendered difficult or even impossible in conditions of swelling, especially in the region of the middle concha, in excrescences or projections of the septum. For the details of sounding the various accessory cavities see later in the respective chapters.

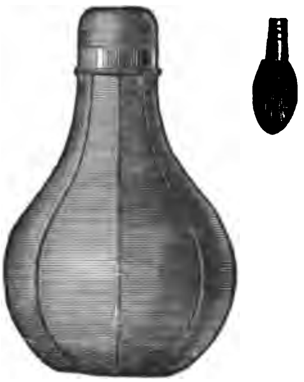

Fig. 14.—*Politzer's* bag with olive-shaped nozzle.

Deflation of the Nose.—In certain diseases of the accessory cavities some authors have recommended deflation of the nose, in order to aspirate the secretions. The simplest method is the so-called *negative politzerization* described by *Seifert*. (See Figs. 14 and 15.)

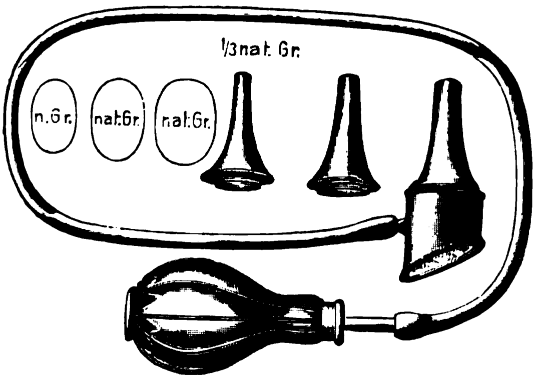

Fig. 15.—Nasal aspirator (*after Schneerson*).

The patient takes a moderate mouthful of water, the nozzle of the compressed *Politzer's* bag is inserted, air-tight, into one nostril, and the nose itself compressed. While the patient swallows with the mouth closed, the bag is allowed to expand. *Sondermann*, *Leuwer*, and *Schneerson* have constructed special nasal aspirators which are introduced tightly into the nostril. The patient is requested to intonate a long-drawn *i* or *k* or a high-pitched *o*, which causes the soft palate to press against the posterior pharyngeal wall and so to shut off the nasal cavity from the pharynx. In my opinion, *Schneerson's* apparatus, which is only a modified *Siegle's* aural speculum, admits the best view of the nasal cavity. The utility of this method is doubted by other authors, who point out that the rarefaction of the air in the nose has an aspirating effect only if the accessory cavities were possessed of flexible walls and were in communication with the outer air by a second opening.

According to *Vohsen*, deflation of the air in the nose causes only hyperaemia and increased swelling, and produces therefore just what is not wanted—obstruction of the openings. Therefore he recommends air pressure; for instance, through air-douching (Politzerizing); whereby currents are produced in the accessory cavities which are capable of carrying with them the secretions. Owing to the uncertainty of all these methods which have now been described,—*Vohsen* certainly underrates the danger to the middle ear,—one should for the present time be satisfied with the usual diagnostic and therapeutic methods, and only in exceptional cases resort to deflation or compression of air.

POSTERIOR RHINOSCOPY (Examination from Behind).

This consists of inspection and palpation respectively of the parts situated most posteriorly, viz.—the naso-pharyngeal space.

For posterior rhinoscopy a tongue-depressor and a small plane mirror are required; the latter fixed at an obtuse angle on a long handle. (See Fig. 16.)

Light is reflected directly on to the soft palate, and whilst with the left hand the tongue is carefully depressed, with the right hand the mirror, previously warmed, without touching the mucous membrane anywhere, is introduced to the left or right of the uvula as far backwards as the posterior pharyngeal wall, in such a way that the mirror looks upwards and forwards.

Mucous bubbles which may interrupt the view may be destroyed by the mirror or be burst by the current of exhaled air.

Examination, however, succeeds only if the velum palati depends flaccidly; for at the moment when it is lifted the view is blocked. It is therefore necessary to direct the patient to breathe quietly and slowly, or that he holds the breath as long as possible, or that he abruptly expirates through the nose; the latter advice, however, is more easily given than followed. Sometimes there remains nothing else but to hook forward the soft palate by means of a palatine hook. (See Fig. 19.) Unfortunately patients are often so sensitive that even the depression of the tongue or the mere touching of the palatine arch excites retching; hence, avoid during the introduction of the mirror any touching of the sensitive palatine arch or root of the tongue, and keep always close to the uvula, which is always insensitive. There is, however, the disadvantage that if one touches the uvula the mirror is smeared and obscured by mucus. In sensitive patients local anaesthesia might be resorted to; but one should, if possible, perform posterior rhinoscopy without such an aid.

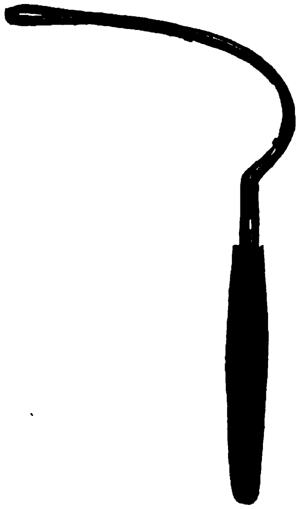

Fig. 16.—Tongue-depressor (*after Fränkel*).

The beginner will doubtless have many difficulties to overcome; but one will nearly always succeed by patience and practice, and, last but not least, by calming the patient, be he adult or child.

In young children and in cases where all love's labour is lost, one should not lose much time, but insert the thoroughly cleansed index-finger behind the velum palati into the naso-pharyngeal space, and palpate quickly the vomer, choanae, and roof. (See Figs. 18 and 21.)

The patient being seated on a chair, the left arm of the examiner is passed behind the patient's head, which in this manner is kept steady, and the lower lip is pushed over the teeth so as to prevent one being bitten; or the cheek may be impressed between the two jaws. (See Fig. 20.) The nail of the examining finger must, of course, be cut short; in spite of this, a little bleeding often occurs.

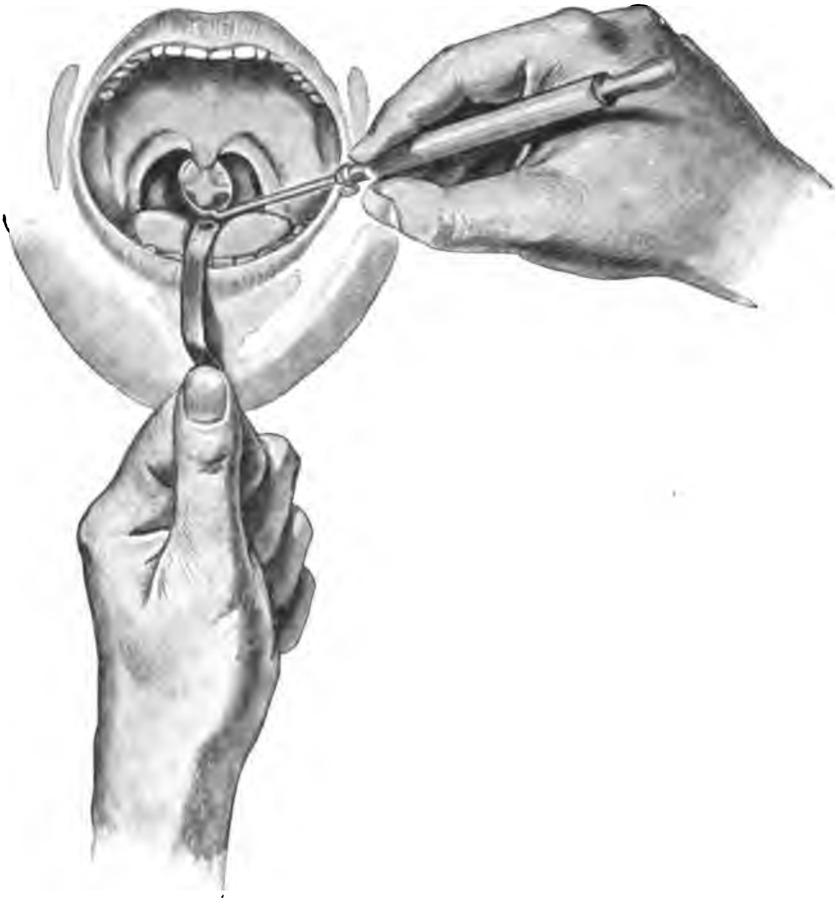

Fig. 17.—Posterior rhinoscopy represented from in front.

This digital examination of the naso-pharynx is somewhat brutal, and should be performed only in cases where it is not possible to make a diagnosis by any other method. Whether

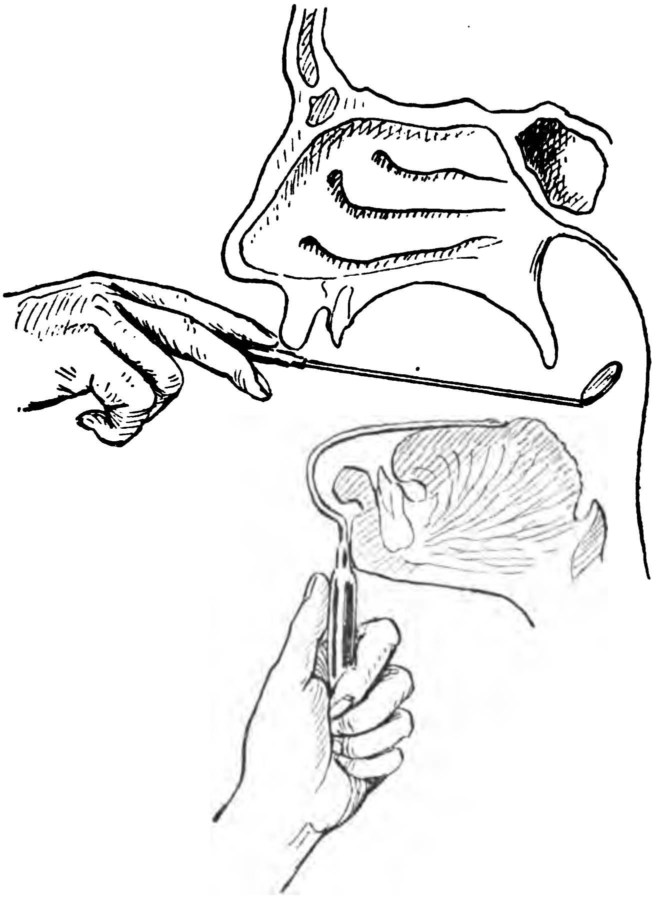

Fig. 18.—Posterior rhinoscopy schematically represented from the side.

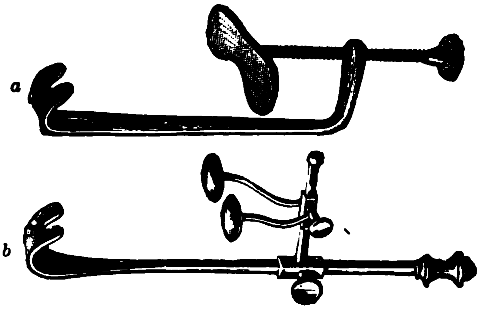

Fig. 19.—Palatine hook: (*a*) after *Krause;* (*b*) after *Schmidt.*

palpation always gives a good result where it is applied may be questioned. Nevertheless it is perhaps the best way of examining tumors in the naso-pharyngeal space with regard to

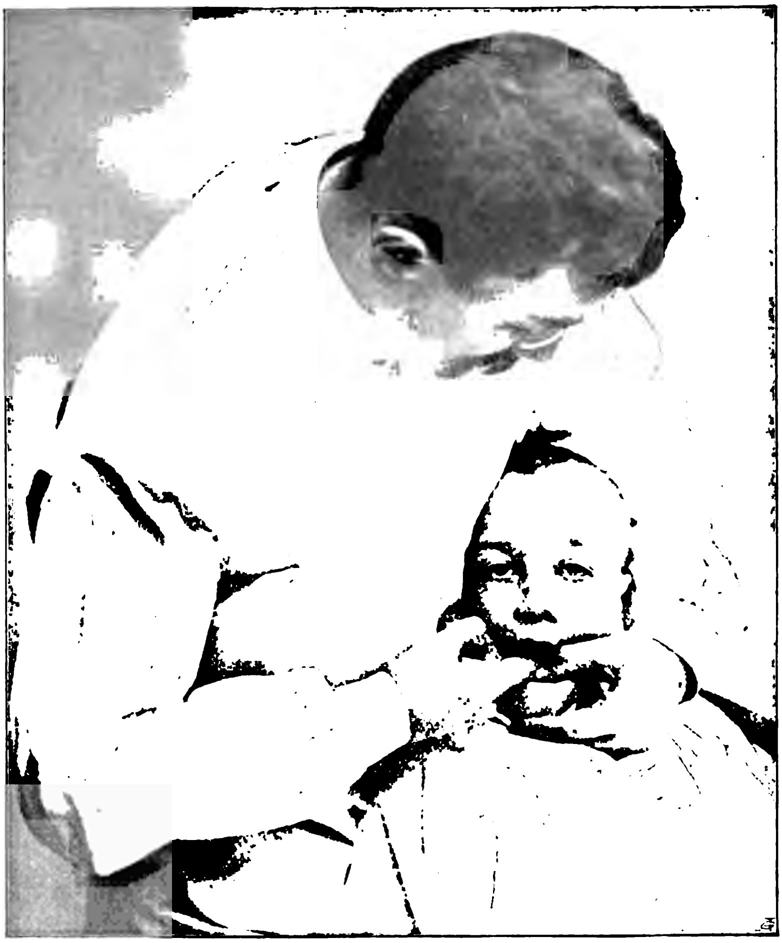

Fig. 20.—Digital examination of the naso-pharyngeal space: Introduction of the index-finger into the patient's mouth.

size, seat, and consistency. In very obstinate children a gag might be used.

Orientation in the post-rhinoscopic image, owing to the smallness of the mirror and to the great foreshortening of the parts, is not easy. The mirror has to be rotated and shifted in order to get a complete survey.

First one inspects the posterior edge of the septum and choanae, then the posterior ends of the three conchae (turbinals), the middle being the most conspicuous. If, now, one has brought into view the lower concha, which is only partially visible, it needs

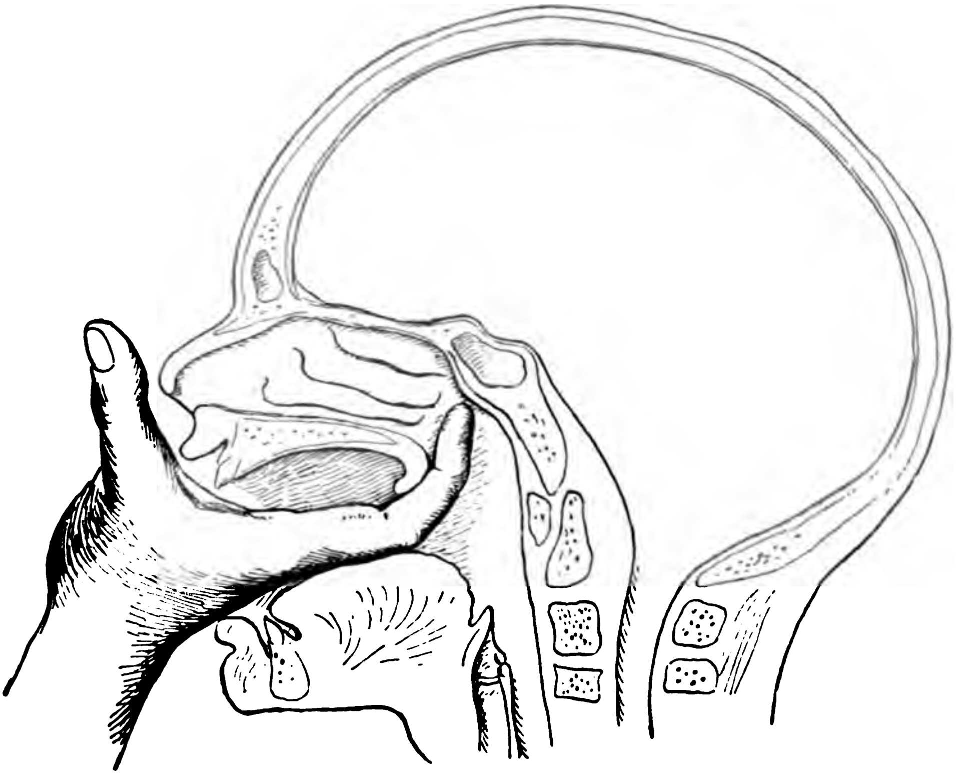

Fig. 21.—Digital examination of the naso-pharyngeal space: Palpation of the space schematically represented from the side.

only a slight lateral rotation of the mirror in order to see the projecting pad of the tube and its ostium. Behind and outside the tubar pad, lying in shadow, *Rosenmüller's* fossa may be seen.

By raising the hand upwards the mirror is rotated horizontally and the roof of the naso-pharyngeal space may be seen as a plane; or if the adenoid tissue is hypertrophied, as a fissured, irregular, lumpy mound, which very often shows, in its midst, an opening called the pharyngeal bursa or the *recessus pharyngeus medius.*

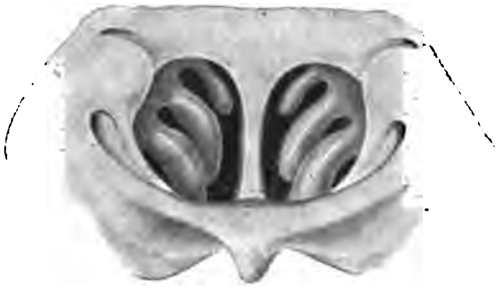

Fig. 22.—Post-rhinoscopic image.

If the mirror is then raised forwards, the posterior surface of the velum and the uvula can be inspected. (See Fig. 22.)

TRANSILLUMINATION.

Transillumination is only required for the examination of the accessory cavities. (See Fig. 23.)

In order to transilluminate the maxillary cavity, a specially constructed incandescent-lamp is inserted into the patient's mouth in a dark room. The examiner asks him to close the lips and the current is then turned on. Then both cheeks appear alight with a red glow, and the pupils also appear red, and the patient himself experiences a sensation of light. If the wall of the cavity is thickened or if the cavity contains an abnormal secretion, the corresponding side of the face remains dark.

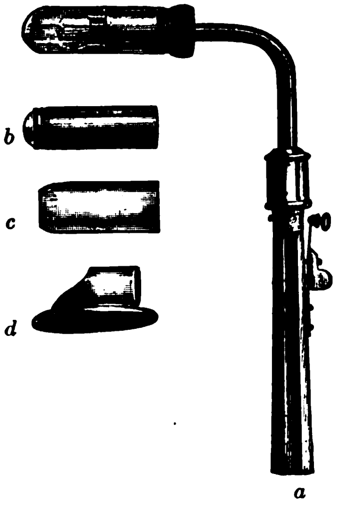

Fig. 23.—Lamps for transillumination (*after Warnecke*): (*a*) Handle with interrupter and lamp; (*b*) magnifying lens; (*c*) india-rubber sheath; (*d*) spatula.

In order to transilluminate the frontal sinus, the lamp, fitted with the magnifying lens and encased in its india-rubber sheath, open in front, is pressed against the inner angle of the orbit.

Examination by *Röntgen* rays is not often required, save for foreign bodies hidden in the nose or its accessory cavities or for a tumour.

ROUTINE OF EXAMINATION.

ANAMNESIS.

1. *Heredity.*

2. *General conditions of life,* occupation, age, etc.

3. *Previous diseases,* especially of the nose.

4. *Present disease.* Duration. Course. Etiology: chill, cold, trade diseases (inhalation of dust, vapours, etc.), bad habits, medicaments (iodine, bromine, arsenic, etc.); infectious diseases—for instance, influenza, acute exanthems, diphtheria, typhoid, pneumonia, tuberculosis, syphilis, and other diseases; injuries and previous treatment.

5. *Subjective Symptoms.*—(*a*) *Pain.*—In diseases of the nose, and still more in those of the accessory cavities, headache of various characters is a very common symptom; sensation of fulness, throbbing, stabbing, continuous or paroxysmal in attack. The pains are localized by the patient in various places. Neuralgic pains in the various areas supplied by the trigeminal nerve and periodical paroxysms of pain, may indicate disease of an accessory cavity.

(*b*) *Psychic Disturbances.*—Depression of mind—lassitude, forgetfulness, and similar symptoms may be very pronounced in obstruction of the naso-pharyngeal space and diseases of the accessory cavities.

(*c*) *Obstruction.*—The sensation of nasal obstruction is often simulated by abnormal dryness of the mucous membrane. On the other hand, narrowing of the nasal cavity through deviation or projections of the septum; swelling of the mucous membrane; new-growths, such as polypi, adenoid vegetations, and abnormal secretions, cause obstruction. The obstruction may jump from one nostril to the other, or shows itself if the patient, coming from outside, enters a warm room. This is due to the changes in the condition of swelling of the cavernous tissue, which is most marked in nervous diseases. The patient calls such a long-standing obstruction of the nose "stuffy-nose." This mostly means that the obstruction is persistent.

(*d*) *Sneezing.*—In diseases of the nose, especially in nervous patients, sneezing as a reflex is very common.

(*e*) *Disturbances of the sense of smell* are present in various degrees. Sometimes the patient himself perceives an unpleasant odour—cacosmia—which is usually a sign of disease of an accessory cavity.

STATUS PRAESENS.

External Nose.—*Inspection.*—Shape of the nose; broadening of the bridge of the nose, for instance, through new-growths, polypi, malignant tumours; "saddle nose" in defects of the osseous or cartilaginous structures due to syphilis; deviation of the nose, due to deviation of the septum near the introitus, or new-growths on one side; swelling or discolouration, for instance, through haemorrhage, boils, dilatation of the blood-vessels, especially in the skin of the upper lip, which in children very often is inflamed, red, and swollen as the result of nasal catarrh; eczema of the introitus nasi.

Palpation.—Consistency; tenderness, for instance, in boils, abscess, inflammation.

Nasal Cavity.—(*A*) *Examination from in Front.*—It is as well to begin with the examination of the nose with regard to its air-permeability, because we can complete the examination more readily, then to investigate for foetor, and to finish with rhinoscopy, having examined the function of smell.

(*a*) *Test of Air-permeability.*—Nose-breathing is entirely obstructed in atresia of the introitus or choanae (anterior and posterior nares); it is more or less diminished if there is stenosis only. Stenosis may occur at any part of the nose from the introitus to the naso-pharyngeal space (formation of scabs at the introitus, inspiratory drawing-in of the alae nasi, deviations and excrescences of the septum, hypertrophy of the conchae, especially the lower turbinal, catarrh of the mucous membrane, scabs, tumours, foreign bodies, adenoid vegetations).

If we find the permeability reduced, the following symptoms can usually be noted:

1. *Stenosis-bruit,* to be tested for on each side; it might be unilateral or bilateral.

2. *Mouth-breathing.* The mouth is kept open, also during sleep (snoring); the expression of the face is dull (stupid). In long-standing mouth-breathing, especially in children, certain changes can be noted in the oral and naso-pharyngeal cavities: viz., shortening of the upper lip, dental caries, high vaulted palate, hypertrophy of the tonsils, pharyngeal catarrh (see below). In infants stenosis of the nose prevents suckling and renders feeding difficult.

3. *Alteration of voice.* The voice sounds thick and repressed (rhinolalia clausa); this is found usually in cases where the posterior region of the nasal cavity or naso-pharyngeal space is obstructed (for instance, by adenoids).

In contradistinction is *rhinolalia aperta,* which is so produced that in speaking and singing too much air escapes through the nose, owing to incomplete shutting-off of the nasal from the oral cavity, for instance, in paralysis of the soft palate. This condition is popularly termed "talking through the nose."

4. *Disturbance of speech.* Constant mouth-breathing alters the speech by rendering it indistinct (stammering), and is sometimes the cause of stuttering.

(*b*) *Ascertaining of Nasal Foetor.*—Bad odours from the nose must be sought for on each side separately. It is very conspicuous in ozaena ("stink nose"); in ulcers and retention of secretion due to foreign bodies, calculi, or suppuration in accessory cavities.

On examining, one must see that the mouth is kept shut in order to exclude mouth-foetor (foetor ex ore) from concretions in the tonsils or carious teeth, etc.

(*c*) *Anterior and Middle Rhinoscopy.*—Inspection of the various parts of the internal nose, introitus septum, floor, conchae (turbinals), posterior pharyngeal wall; especially important are: *changes in the secretion,* quantitative and qualitative.

Quantitative changes: hypersecretion in acute or chronic inflammation of the nose or its accessory cavities; diminished secretion, usually in chronic processes of atrophic character; periodic discharge of pus in suppuration of the accessory cavities.

Qualitative changes: the secretion is watery, and if copious, is called *hydrorrhoea nasalis,* in vasomotor lesions of the nose; or it is glairy, mucous, or muco-purulent or purulent, and inclined to exsiccation. A purulent secretion is found in various conditions; for instance, in suppuration of the accessory cavities; scabs are formed chiefly in atrophic conditions. Sometimes the secretions are bloody or blood-stained; for instance, in ulceration, or in atrophic disease of the mucous membrane. Fibrinous secretion is found in diphtheria, fibrinous rhinitis, and after caustic applications.

It must be further ascertained whether the discharge is *unilateral*—as it is mostly in disease of the accessory cavities and in cases with foreign bodies—or *bilateral.*

Finally, the *place* from which the pus is derived must be noted, although it does *not always* admit of absolute diagnosis. Pus in the hiatus semilunaris, in the anterior part of the cavity and floor of the nose is derived from the maxillary or the frontal sinus or anterior ethmoidal cells, or from several of these cavities. Pus in the olfactory chink (rima olfactoria) and in the posterior part of the nasal cavity or naso-pharyngeal space and on the posterior pharyngeal wall, comes usually from the posterior ethmoidal cells or the sphenoidal sinus. (For details see special section.)

Examination is completed by:

1. Probing (sounding); mobility and consistency of the parts concerned; tenderness of the mucous membrane; polypi; ostia of accessory cavities.

2. Anaesthetizing and anaemizing (artificial anaemia).

(*d*) *Examination of the Sense of Smell.*—This will be necessary only in a case where the patient himself complains of disorder of his sense of smell; and where certain conditions are present which lead us to suspect such a lesion, as, for instance, cerebral disease. Each side of the nose must be tested separately; and the sense of taste must also be noted.

(*B*) *Examination from Behind.*—*Posterior rhinoscopy,* and if this is impossible, or has to be supplemented:

Palpation of the Naso-pharyngeal Space.—There are to be noted: Adenoid vegetations; tumours; ulcers in the naso-pharyngeal space; atresia of the choanae; collections of pus; alterations of the posterior extremities of the conchae, etc.

(*C*) *Transillumination.*—1. *The Maxillary Sinus* (*Antrum of Highmore*).—If one half of the face below the infraorbital margin remains dark, if the pupil does not appear red, and the subjective sensation of light is missing, a collection of pus in the respective sinus is probable; *but only if there are also other characteristic symptoms of suppuration of that cavity.* The fact of remaining dark *per se* proves nothing, because it might be also produced by asymmetry of the walls of the antrum, by tumours, and hypertrophy of the cavity, by polypi; and, last but not least, by tampons in the cavity of the nose (*M. Schmidt*).

Still less reliable is bilateral darkness, which is to be explained by the thickness of the facial bones; on the other hand, transparency does not disprove suppuration of the sinus.

Transillumination, therefore, of the maxillary sinus is a very good aid to diagnosis, *but does not assure it.*

2. *Frontal Sinus.*—Transillumination of the frontal sinus is still less valuable; it does not even show us the limits nor the nature of the contents of the sinus.

Other Organs.—1. *Eye.*—Diseases of the nose and its accessory cavities are liable to implicate the eye, either by direct continuity of the inflammation to the orbit, or by transmission of germs by lymph and blood-vessels or by mechanical influences on one or other part of the orbit.

One finds lachrymation (epiphora), due to obstruction of the ductus naso-lachrymalis; conjunctivitis; impairment of vision; contraction of the visual field; asthenopia; and in severe cases abscess of the orbit with iritis, exophthalmos, etc.

2. *Ear.*—Diseases of the nose are often the source of affections of the ear, especially in children, because, owing to the greater width and shortness of the Eustachian tube, the germs are more easily transmitted to the cavum tympani than in adults. If the air-way through the nose is obstructed—for instance, by catarrh, swelling of the conchae, adenoids, etc.,—occlusion of the tube may result, which in the end leads to rarefaction of the air in the tympanic cavity and corresponding retraction of the tympanic membrane. Consequently, on the rarefaction of the

air in the cavum, transudation takes place into it, as we see it in cases of hydrops ex vacuo. In other cases suppuration occurs. Hearing is impaired either temporarily or permanently.

3. *Upper Air-passages.*—Inflammations of the pharynx, larynx, and trachea are often sequelae of diseases of the nose and its accessory cavities. This is due to the secretions flowing down and irritating these passages (descending catarrh).

4. *Teeth.*—Obstructed nasal respiration often causes caries of the teeth, for it fosters the invasion of the mouth by bacteria. *Mancioli* found that in mouth-breathers the upper incisors and lower molars are chiefly affected.

5. *Brain.*—Meningitis and abscess of the brain may occur in inflammations of the accessory cavities through inflammation extending into the skull. It is not yet quite certain whether the infection of the contents of the skull ensues as a result of pervasion through the bone, or by the bones themselves being first infected. According to *Weichselbaum*, cerebrospinal meningitis is not infrequently to be traced to rhinitis.

6. *Stomach.*—Diseases of the stomach are sometimes simulated by the frequent vomiting due to abnormal irritability of the pharynx. Unpleasant sensations and a bad taste in the mouth, and morning sickness, are often symptoms of suppuration in the accessory cavities (*Hajek*).

7. *Reflex Disorders.*—The nose plays a great part as an organ exciting reflexes in other organs, especially in nervous patients. In particular, *asthma may be excited.*

We have tried in the above to give a scheme which may suit most cases. It is, of course, not necessary to emphasize the fact that in not every case of nasal disease is there need of such a thorough analysis with regard to arriving at a diagnosis. It might be sufficient in many cases to assure oneself of a few facts; and external inspection with anterior rhinoscopy will accomplish all that is required. In other cases it is sufficient to ascertain the more important points.

GENERAL THERAPY.

GENERAL MEASURES.

Treatment of nasal diseases can only be considered as appropriate if one takes into consideration all the manifold relationships between affections of the nose and those of the whole body. A great number of nasal diseases originate in general disorders, or are in connection with diseases of other organs or groups of organs. A solely specialized treatment, therefore, is not feasible, neither in the nose nor in other portions of the air-passages. Our attention should always be directed to an eventual primary cause or to a general disease, and our treatment has to be directed accordingly. The nose does not require any special treatment in many cases. We very often succeed by general measures (see below).

LOCAL TREATMENT.

(*a*) **Cleaning the Nose.**—*Blowing.*—The simplest way to clean the nose is blowing it "*à la paysan.*" The patient shuts one nostril—eventually the physician has to do it—and blows through the other nostril into a receptacle or handkerchief. This method, of course, is possible only where the nasal cavity is not too narrow and the secretion not too dry. Small children cannot blow the nose; and here one can help matters by inserting the olive-shaped nozzle of the india-rubber bag used for Politzerization into one nostril, and expel the air by repeated sharp compressions whilst the child breathes quietly. The secretions are thereby expelled through the other nostril or into the pharynx.

Wiping Out the Nose.—If the patient is not too sensitive and the secretions are not too massive, one can use a sponge-holder dressed with cotton-wool, or even forceps, in the same way. This method can be made more easy by previous local anaesthesia and anaemia.

Gottstein's Tamponade.—If the secretions are desiccated, a plug of cotton-wool is put into the nose and left there for some time. If then the plug is removed, the scabs adhere to it, or they are rendered so loose that their removal becomes an easy matter. This method is called "*Gottstein's* tamponade," and it acts reflexly by inducing secretion. It is well to moisten the plug with a little fluid paraffin.

Douching.—The sniffing up of fluid or douching the nose with large syringes under strong pressure should be avoided, in order to prevent the fluid from penetrating into the ear or accessory cavities. A nose-spray is more safe, but is also more complicated. (See Fig. 24.) In my opinion, douching with so-called nose-douches seems perfectly satisfactory in cases where we desire only to clean the nasal cavity and naso-pharyngeal space. These nose douches are pear-, funnel-, or bell-shaped glass vessels of suitable size, with a short inlet and a longer bulb-tipped delivery nozzle. (See Fig. 25.) I admit that in all cases the secretions are not removed, but they are at least moistened and loosened, and thereby their removal is made easy. In exceptional cases I allow the patient to use an irrigator, which, however, must not be hung high up, so that no pressure can be exercised; or instead of this, a small syringe may be used.

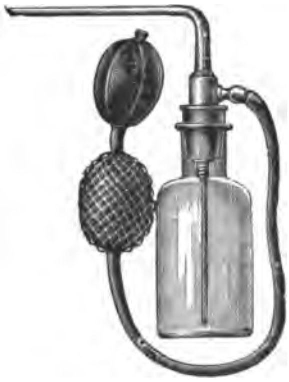

Fig. 24.—Nose-spray.

Precautions to be taken in douching the nose: 1. Use no cold fluids or strong solutions, but tepid, indifferent fluids of 25° to 30° C., and weak solutions of common salt (sodium chloride), bicarbonate of soda, borax, or boric acid. Of borax, use one-half teaspoonful to half a pint of water; and of boric acid, in solution 1 or 2 per cent.

2. The solution should be run in through the narrower nostril, so that it can escape through the wider nostril.

3. The patient should bend his head a little forward so that the solution flows across the choanae into the other nostril.

4. No pressure should be exercised, and the flow should be even, in order that the soft palate remains contracted and the naso-pharyngeal space shut off from the mouth.

5. During douching the patient should not be allowed to

swallow, but breathes quietly through the mouth. If a movement of swallowing is excited, douching should be interrupted.

6. The patient must be forbidden to blow the nose after douching until the fluid has entirely drained away. Then he may gently blow each side separately.

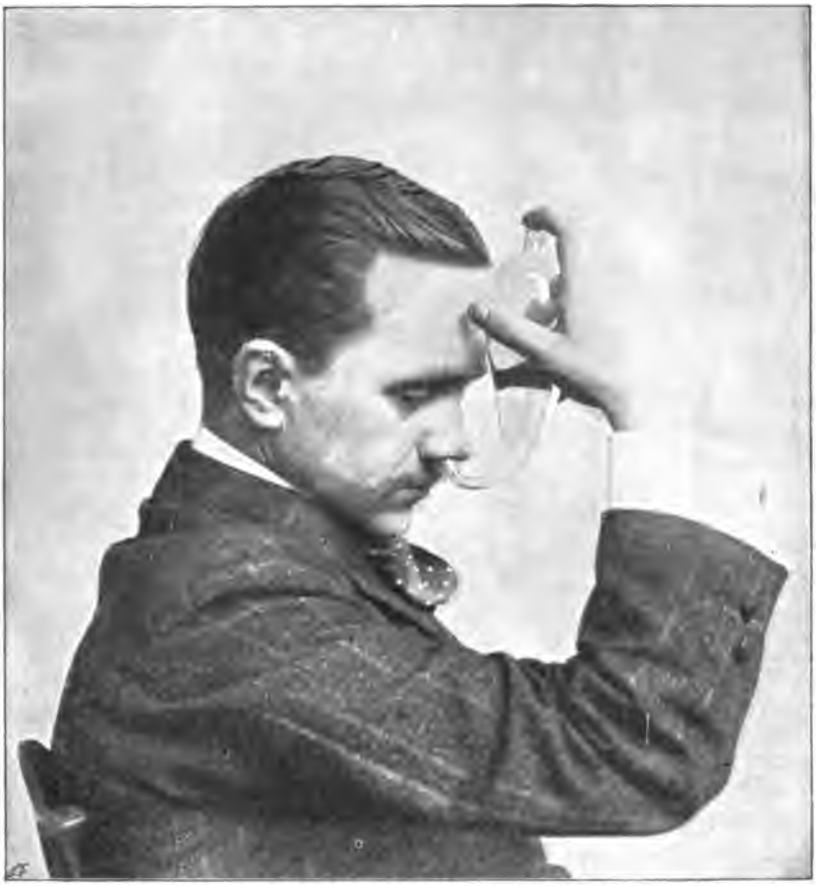

Fig. 25.—Nose-douching with *Starke's* douching apparatus.

7. After douching in cold weather the patient shall stay in the room for a time.

8. So as not to occasion headache, the jet of fluid, if a syringe be used, should not be directed towards the roof of the nose (upwards), but horizontally backwards.

(b) Treatment by Drugs.—Medicaments are introduced into the nose either as fluids, by painting, brushing, instillation, and massage; or as powders, by insufflation; or as "snuffs" (sniffing); or as vapours, by inhalation. For the purpose of painting, a probe or sponge-holder may both be used, dressed with cotton-wool, and the same also for massage. To perform the latter, the mucous membrane is rubbed in short rapid movements which should only be executed from the wrist, after previous cocainization, however (vibration massage). The hand may be replaced by an electro-motor. For instillation, an ordinary eye-dropper can be used. "Snuffs" can be sniffed up through each nostril separately. Powders can be insufflated by means of an insufflator. (See Fig. 26.) Volatile medicaments may be poured into the hollow of the hand or onto blotting-paper or the pocket-handkerchief, and sniffed up; or by means of prepared plugs of cotton-wool placed in the nostril; for instance, formaldehyde or menthol plugs.

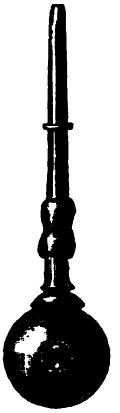

Fig. 26.—Insufflator.

For brushing I use solutions of silver nitrate 1 to 10 per cent. and iodine (℞: iodi puri, 0.2 to 0.6; potassii iodidi, 2.0 to 3.0; glycerini pur., 20.0; ol. menth. pip., gtt. ij.).

For massage of the mucous membrane I use the above iodine solution, or a mild ointment such as vaseline, boric-vaseline, lanoline, and byroline.

Instillations are needed only in the cases of small children, and here I use liquid paraffin, of which I put into each nostril one to three drops whilst the head is bent somewhat backwards.

Powders are insufflated only after operative manipulations, and even then seldom (see below).

"*Snuffs*" are supposed to excite irritation of the mucous membrane or to cause shrinking of the same. For irritation, I use borax or boric acid; for shrinking, I use menthol or cocaine.

Cauterization.—For cauterization, silver nitrate and perchloride of iron (both drugs in weak solution), and also chromic acid and trichloracetic acid (both in strong solution), are used. To apply perchloride of iron and trichloracetic acid a small probe can be used, the end of which is dressed with a small piece of cotton-wool and then lightly dipped into the caustic. (See Fig. 27.) Caustic points of lapis infernalis and chromic acid are fixed to a probe or caustic-holder. The probe is heated over a flame till it is red; it is now brought into contact with the caustic point. When it has again become cooled, a small quantity of the medicament will have adhered to the probe, forming a white bead thereon. Chromic acid is not so easy to manage. The probe or causto-phore is moistened and some of the chromic acid crystals are caught thereon and then rendered molten at some distance from the flame. When cooling down, the chromic acid forms an amorphous mass on the probe, of a sealing-wax red colour. If the chromic acid be heated too much, black chromic oxide is formed, which is ineffective. Chromic acid is very hygroscopic, and must therefore be kept well protected from the air. Liquefied chromic acid, however, is also a good caustic, and can be used by means of small cotton-wool swabs.

Perchloride of iron and chromic acid stain the skin yellow. Lapis infernalis and trichloracetic acid leave white sloughs. The caustic should always be used in small quantity and be placed on the exact spot which it is required to cauterize. Lapis infernalis and chromic acid should not be allowed to form large beads because they break off very easily. Before cauterizing, local anaesthesia is required, and afterwards the superfluous caustic must be swabbed off.

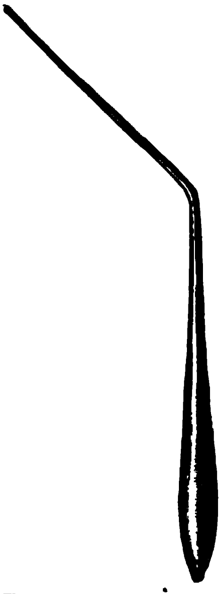

Fig. 27.—Probe for cauterization.

(c) **Operative Treatment.**—1. *Local Anaesthesia.*—Almost all intra-nasal manipulations are, at the present time, performed under local anaesthesia. For this purpose, cocaine, 10 to 20 per cent. solution, or alypin solution, 10 per cent., are used, having previously anaemized by the application of adrenalin. The use of these drugs for the purpose of examination has been already described (page 21). For treatment, the aforesaid description (on page 21) holds good. In some cases, especially in operations on the septum, *Schleich's* method of local anaesthesia might be resorted to. After having cocainized the site of injection, *Schleich's* weak solution is injected under the mucous membrane until it is well raised like a bleb. (*Schleich's* weak solution: Cocain. mur., 0.01; morph. mur., 0.002; sodii chlorid., 0.2; aq. dest. steril., ad 100.0; sol. acid. carbolic. [5 %], gtt. ij.) *Baumgarten* is satisfied with only a physiological solution of sodium chloride, to which he adds eucaine (sodii chlorid., 0.6; eucaine, 0.2; aq. destill. steril., 100.0), and uses a curved cannula for injection. After operation firm tamponade will be required in order to prevent after-haemorrhage.

2. *General anaesthesia* is not often required in intra-nasal operations, but it might be useful in obstinate children who are not easily controlled; for instance, in order to remove a foreign body from the nose. I use *chloroform* by the usual method of dropping it on a mask. The anaesthesia should only be light, *so that the coughing reflex should not be abolished,* in order to prevent aspiration of blood or secretions. For short operations I use ethyl bromide (aether bromatus purissimus). A large quantity is poured on to a mask, which is covered with an impermeable material (for instance, "*Mosetig's* Battiste"), and it is then pressed onto the face; 10 to 15 grammes are sufficient, sometimes more. Unconsciousness occurs after one-half to one and one-half minutes and lasts about two to three minutes; hence quickness in operation is desirable.

More recently ethyl chloride has been recommended; the warmth of the palm suffices to expel ethyl chloride from the capillary opening of the tube onto the mask covered with six or eight layers of gauze.

3. *Instruments.*—Various instruments are used for nasal operations. They and their uses will be described in the special section under their respective chapters. Here, it is only necessary to say a few words on galvano-cauterization and electrolysis.

Galvano-cauterization.—The source of electricity may be a galvanic battery, an accumulator, or the main-supply. For the treatment of the nasal cavity pointed and flattened burners, and snares of platinum or steel wire are used. (See Fig. 28.) As a handle I prefer *Kuttner's* universal instrument. The burner and snare should not be made hot before they are placed on the exact spot. In order to prevent bleeding the burner should not be heated to more than a dull red and should be lifted from the mucous membrane whilst still red. White heat does not act as a styptic. Touching the skin of the vestibule is a mistake and should be avoided. Galvano-cauterization is a very efficacious means of destroying tissue, and has the advantage of acting only on a particular spot and also as a good styptic. These advantages are so conspicuous that any discomfort experienced afterwards by the patient are hardly to be considered; and they are so fascinating that galvano-cauterization has perhaps fallen into abuse. Nowadays a certain disinclination can be observed with regard to galvano-caustic over-enthusiasm, which we can only approve. Galvano-caustic and other operations in the nose should be reduced to the utmost minimum which will still preserve the physiological protective powers; provided that they have not already been lost by previous diseases.

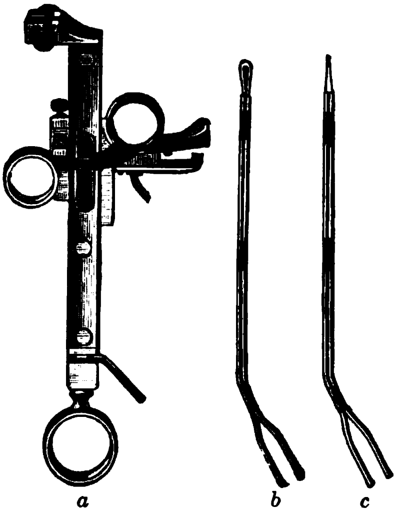

Fig. 28.—*a*, *Kuttner's* galvano-cautery, universal handle; *b*, flat burner; *c*, pointed burner.

Electrolysis is relatively seldom utilized, because of its slow effect. If a platino-iridium needle be inserted into the tissues and the current turned on, then with a crackling noise at the positive electrode (anode) oxygen is given off, and at the negative electrode (cathode) hydrogen is evolved. Water is abstracted from the tissues and decomposed into its component elements by the galvanic current. At the anode the tissue is coagulated, owing to the oxidation, and at the cathode it is liquefied. Thus the tissue is slowly destroyed; and the destruction is proportional to the strength of the current and to the duration of its action. For this purpose single or double needles are used (unipolar or bipolar methods). (See Fig. 29.) Double needles are more effectual. In the unipolar method the positive button electrode is usually placed on some part of the skin of the body and the negative electrode is inserted into the particular site in the nose. In the bipolar method both needles act together. The site concerned is previously anaesthetized. The intensity of the current has to be measured according to the sensitiveness of the patient, and the current slowly turned on and off. The single sitting should last from five to ten or twenty minutes; but very often a whole series of sittings are required, which take place at intervals of a few days.

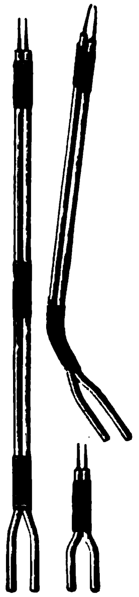
Fig. 29.—Double needles for electrolysis.

HYGIENE AND PROPHYLAXIS.

In the wonderful development of modern medicine we see that, not only medical treatment, but hygienic and dietetic factors are more and more taken into consideration, and, hand in hand with them, more stress is laid on prophylaxis. This makes it our duty to become acquainted with all the factors which assist the organ (the nose) in maintaining its functional powers, and to protect it from damage. The public themselves require advice from the specialist as to hygiene and prophylaxis, and our entire services are sometimes solely completed therewith. Owing to the intimate relation between the nose and other organs, we have to direct our attention and care frequently, not to the nose alone, but to the whole body. The specialist must always be a complete physician; he should never regard *partem pro toto* (the organ for the organism).

The most important question here concerned is "*hardening.*" The more "hardened" a man is, the less liable is his nose to the danger of "cold"; and likewise the upper air-passages, which are so much exposed to every possible injurious influence.

How a "cold" occurs is not always easy to ascertain. Sometimes it is by a sudden general cooling down of the body, and in other cases the "cold" hits only a certain part of the body: *e. g.*, the head, the neck, the feet, etc. "Hardening" has to be adapted to the individual. In infants and old persons it is better to abstain; whilst in children from the second year upwards the adaptation to weather changes must be carefully regulated. This can be done by the regular application of cold water, movements, and suitable clothing.

At the beginning of the "hardening" process tepid water should be used, about 20° C., and gradually diminish the temperature daily by half a degree until one has arrived at a temperature of 10° C. The method, however, must be directed according to conditions and individual needs. I have seen very good effects from cold frictions, which possess not only good results, but also are very convenient and cheap. For this method linen or Turkish towelling soaked in cold water and wrung out is necessary. A large sponge might also suffice. The friction is best done in morning on rising. Sometimes frictions can be ordered twice daily, in the morning and evening. It might be limited to the chest and back, or to neck and limbs, or the whole body, in sections, can be rubbed down. Having completed the cold wet friction, a dry friction must follow. All the hardening by hydropathic procedures, more especially in children, must be performed gradually and directed according to individuality.

Hydropathic hardening is greatly assisted by bodily exercise, such as gymnastics, rowing, swimming, tobogganing, etc., and by suitable clothing. Too warm clothing is not desirable. It is not the place here to enter into consideration of the controversy of clothing faddism, or to decide whether wool, cotton, or other materials be preferable. *B. Fränkel* and *M. Schmidt* are not strong advocates of the woollen regimen of clothing, and the former speaks of wool as a "new cause of enervation." Woollen material as underwear is objectionable and should be allowed only where the skin is readily prone to perspiration. On the other hand, it is well to recommend that a netted vest of cotton or silk should be worn beneath the shirt. In any case the neck should be left free.

Systematic hardening is only possible in rooms of suitable temperature. The disposition to "colds" and catarrhs of the upper air-passages *is greatly increased by overheated apartments.* The temperature of living-rooms should be 16° to 19° C. The bed-room should be temperate. But here, as in many other matters, habit is our master. There are people who sleep even during the coldest winter in unheated rooms with open windows; in such cases care should be taken that a draught does not blow directly onto the bed.

The air should be free of dust and smoke and should possess a certain amount of humidity, so that the nose is not hindered in its function of cleaning and moistening the inspired air. Clearing of the nasal cavity by blowing the nose should be done through each nostril separately. (See page 37.) Pocket-handkerchiefs should be frequently changed, in order to prevent as much as possible the transmission of bacteria at least.

The nasal cavity is often polluted by the wide-spread, but nevertheless unclean habit of "snuff-taking," and by the custom of ejecting cigarette and cigar smoke through the nostrils; it is not improbable that the sense of smell is injured. As is tobacco, so also is alcohol noxious; and in smokers and drinkers swelling of the mucous membrane can very often be found, which in turn impedes nose-breathing.

The swelling in the cavity of the nose is also apt to give rise to chronic congestion and is liable to cause "copper nose" ("grog blossoms"), which is so characteristic of drinkers. Thus we see that alcohol and tobacco play a great part in the etiology of nasal diseases; hence it should be our endeavor to exclude both where nasal disease or the disposition to such is present.

Professions and trades with malign influences on the nose are numerous. *Röpke* enumerates not less than one hundred and fifty of these, all of which lead to acute catarrh of the nose and the upper air-passages. For these, general rules as to hygiene and prophylaxis are not applicable. At the most, one can only advise the patient so as to reduce the pernicious effects of his particular occupation and to place him in the most favourable circumstances with regard to his respiratory organs, so far as his occupation permits.

SPECIAL SECTION.

I. DISEASES OF THE SKIN OF THE NOSE.*

ECZEMA VESTIBULI.

(Eczema ad introitum nasi. Eczema of the Entrance of the Nose.)

Eczema of the introitus is either acute or chronic. In the acute form it shows various degrees of redness and swelling, with frequently the formation of vesicles, scabs, and fissures. In the chronic form the skin is infiltrated—frequently indurated—and shows cracks and fissures covered with numerous scabs, which might be so thick that the entrance of the nose may be blocked. The process often invades the adjacent parts of the upper lip, which then appears swollen and everted; and the fissures, though they may be small, form a ready entrance for pathogenic germs, viz., erysipelas, boils, sepsis, sycosis, and syphilis.

Etiology.—The eczema in the majority of cases may be traced to the irritation caused by the secretions of the nose itself; as in acute or chronic rhinitis, or from the accessory cavities, or naso-pharynx, especially in cases of adenoid vegetations. It is very often found in scrofulous children.

Course.—The course is often protracted. The patient has frequently to use the handkerchief for the persistent irritation, the painful tension, and the frequently impeded breathing through the nose. The patient picks the nose, thus scratching and separating the scabs, causing yet again fresh irritation. Haemorrhages readily occur, and if infection supervenes, it may lead in very chronic cases to ulceration and perforation of the nasal septum (ulcus septi perforans).

Diagnosis.—The diagnosis, as a rule, is easy. Confusion with lupus vulgaris or syphilis has occurred. Deep fissures or ulcers are probably due to syphilis. The specific character of the affection is only at length discovered by the examination of the whole body.

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* Though numerous affections of the skin of the nose are often only a part of general affections of the skin found elsewhere, and which befall the whole integument, yet a short discussion of them does not here seem superfluous, for the practical reason that patients suffering from those complaints often consult the rhinologist; and secondly, because many of the above-mentioned affections are in more or less intimate relation to the disease of the nasal interior.

Treatment at first must aim at removing the scabs. This can best be done by putting small swabs soaked in warm olive oil or liquid paraffin into the nostrils, where they should remain for a while. The scabs are thus softened, and can then be carefully cleared away. Afterwards, any kind of indifferent ointment should be applied, such as vaseline, or byroline, etc., or in very obstinate cases diachylon or white precipitate. This anointing should be done several times a day, especially after each cleansing, or at least twice a day, morning and evening; and, if necessary, with a small wooden stick, dressed at the end for this purpose with cotton-wool. The patient should be advised to refrain as much as possible from any mechanical irritation, and to exercise extreme care with regard to cleanliness in the treatment. *Fissures* are best treated by a 2 to 10 per cent. solution of nitrate of silver; and in very chronic cases cauterized with caustic points of lapis infernalis. The treatment, of course, must take into consideration the cause of the disease—*cessante causa cessat effectus*.

SYCOSIS VESTIBULI (SYCOSIS; PUS-INFECTION).

Sycosis is an inflammation of the hair-follicles leading to suppuration. It often occurs within the entrance of the nose, where the small hairs, known as vibrissae, grow (*e. g.*, at the anterior nares), and then frequently spreads, in men, over the upper lip. The disease begins with small, hard, red nodules or papules, each being perforated by a hair. These small nodules tend to break down by suppuration, thus forming small pustules, which dry up and are then covered with scabs. By this process the whole upper lip, point and wing of the nose are soon infiltrated, and thus much disfigurement ensues.

Origin.—The origin of sycosis is not always clear. In many cases it is due to the irritation caused by the nasal secretions containing staphylococci. Sometimes an already preëxisting eczema may lead to sycosis in its further course (*Lesser*, *Schech*).

Course.—The course is always protracted, and is retarded by relapses and is sometimes complicated by painful furunculous inflammation. In long-standing cases the hair is usually lost and the skin becomes scarred, due to atrophy of the follicles.

Diagnosis.—The diagnosis at the beginning is easy. Each papule or pustule is then perforated by a hair. Later on, if the hair is lost and scabs are formed and all the parts are infiltrated, thus masking the picture, distinction should be made from chronic eczema, tinea, syphilis, and lupus. In uncomplicated sycosis, there is no such exudation as in eczema or tinea. In lupus and syphilis, ulceration is the main feature.

Treatment consists in gentle epilation of the hair by means of ciliary forceps, in removing the scabs, as we have shown in the foregoing; and afterwards, anointing with white precipitate, or sulphur ointment, or ichthyol. Through the epilation, many pustules are opened, but the others must be incised with a small lancet. The use of the handkerchief should be avoided; and cleansing of the nose should be done by blowing "*à la paysan,*" or, what is more refined, by mopping with cotton-wool.

FURUNCULOSIS VESTIBULI (FURUNCULOSIS; BOILS OF THE ENTRANCE OF THE NOSE).

A boil is an inflammation of the sebaceous glands of the skin, ending in suppuration through infection from staphylococci, and, as a matter of fact, is nothing else than a large acne pustule. It often occurs at the entrance of the nose in persons of unclean habits, who pick their nose. Chronic eczema and sycosis predispose to it. The onset is slow; the patient feels a sort of pricking and often an increasingly painful tension. The skin at the point or wing of the nose swells, reddens, becomes infiltrated, and a swelling may be frequently noticed inside the nose, which shows on the top a yellowish appearance from suppuration. A boil situated at the angle of the entrance often conceals itself and can only be discovered if the patient's head is bent strongly backwards. It may be more easily detected by viewing the introitus in a reflecting mirror held in front of it.

Treatment at the beginning should be to relieve pain by covering the infiltrated parts with vaseline or byroline. It is still better to lay small pieces of cotton-wool soaked with acetate of aluminium (a teaspoon to ¼ l.) over the edge of the nose like a horseshoe. Sometimes I found that the infiltration did not pass on to suppuration, and the inflammation subsided by this method. Usually after a few days the furuncle breaks down, the necrotic matter is discharged, and it soon heals. An incision is mostly unnecessary. The patient should be strenuously advised to protect himself from furunculosis by cleanliness and the avoiding of any irritation.

COMEDONES.

The skin of the nose is a favourite place for comedones, especially in young adults at the time of puberty. They are caused by retention and thickening of the secretions of the sebaceous glands in the blocked and often dilated gland ducts. To the eye they appear as small black points, embedded in the mouths of the follicles, and are often elevated above them. They look like small yellowish worms with a black head, whence came the name of comedo. The black head is due to dirt. Microscopically, horny and fattily degenerated cells are found, together with fat globules and fine hair-roots; and often a parasite, 0.3 to 0.4 mm. long, *the acarus folliculorum.* This acarus is a very harmless though very common parasite, especially in persons suffering from seborrhoea (page 51). It has no connection with the origin of comedones.

Through the irritation exercised by the thickened and retained secretion of the glands, comedones often cause a certain kind of inflammation of the follicles, tending to suppuration. This is then called "acne vulgaris."

Treatment.—Comedones are removed by expressing them with the clean fingers or special instruments. It is useful to previously soften the skin by washing it with soap and warm water. In order to prevent retention and blocking by the secretions various means may be resorted to, all of which serve the purpose of removing the superficial layers of the epidermis and freeing the mouths of the sebaceous glands. Sulphur ointment (precipitated sulphur 3 parts, lanoline 30 parts) is smeared over the respective part at night; and on the following morning is washed off with soap and water or benzine. Of similar effect is the following ointment:

℞.	Resorcini	3.0 to 5.0
	Zinci oxidi	
	Amyli tritici	āā 12.5
	Vaselin. flav	ad 50.0
M. Fiat pasta.		

Or—

R.	Sulphuris depurat	5.0
	Resorcin	2.5
	Vaselin. flav.	ad 50.0

In some long-standing cases corrosive sublimate 1 per cent., externally applied, has a good effect; care must be taken, however, that it does not come in contact with the eyeball. All the above-named remedies should be stopped if the skin shows much irritation from their use. The desquamation may be aided by an indifferent ointment (zinc or byroline). Very obstinate acne may be covered with a mercury plaster. The use of a marble soap (20 %) or sulphuretted and camphorated soap is highly recommended.

SEBORRHOEA NASI.

This affection, which occurs on the nose and forehead, is caused by hypersecretion of the sebaceous glands. The skin has a shiny, fatty appearance, as if it were anointed (seborrhoea oleosa). It is often accompanied by comedones and eruptions of acne.

Treatment can only consist in cleaning and washing the skin. Diluted spirit of wine is very useful for this purpose.

CONGELATIO NASI (FROST-BITE).

Freezing of the nose frequently occurs in anaemic persons. Mostly, the first stage of frost-bite is met with. The nose, especially the point of the nose, is bluish-red; and on pressure, round white patches readily appear, which soon resume the colour of the neighbouring parts. The discolored skin itches, burns, or is painful, and this especially at the transition from a cold into a warmer temperature. In long-standing cases after many relapses—if once frost-bitten it is inclined to become frost-bitten again—the nose is apt to be much disfigured, and, like the hand and foot, liable to chilblains (perniones). Blisters and sloughing, as is so frequent in the case of the ear, is not often met with in the nose.

Treatment consists in the application of a tonic ointment (camphorae trit. 5.00 ad vasel. flav. 50.00), rubbed in at night and before going out. Also an ointment of chloride of calcium is very successful:

℞. Calc. chloridi	1.00
Ung. paraff.	9.00
F. ung.	

A small piece is to be rubbed in morning and evening for five minutes and then covered with impervious material (gutta-percha paper). It is useful to cover the frozen parts every evening for ten to twenty minutes with small pieces of cotton-wool soaked in hot diluted vinegar (1 tablespoon to ½ l.) and after that, apply the ointment. It goes without saying that the general health of the patient is of the utmost importance, and prophylaxis should not be neglected.

DISTURBANCES OF CIRCULATION OF THE SKIN.

(Copper Nose; Erythema; Angeioma; Acne Rosacea; Pendulous Nose.)

The name of red or copper nose is not a scientific one. It means the dilatation of all the small, and smallest, vessels of the nasal skin, whether the conditions be transitory or permanent.

Erythema is a patchy or diffuse redness, caused mostly at the point of the nose by venous or arterial hyperaemia, as a consequence of chronic inflammation of the inside of the nose, of previous frost-bites (vide above), or—and this is the most common—a more transitory *erythema fugax*, due to reflex action of the vasomotor nerves. This fleeting redness is often met with in young women or girls; frequently in connection with menstruation. All kinds of stimuli may produce this redness of the nose, as also transition from cold to room-temperature, mental or psychic agitation, and last but not least, the drinking of hot, but not necessarily alcoholic, beverages. The patients are much distressed by the complaint, as they are often exposed to unjust suspicion. The affection is of a very harmless nature.

Treatment, which is of very little avail, must take into consideration the cause, and endeavour to effect a remedy.

Telangiectasis (naevus) is the persistent dilatation of capillary blood-vessels, and is found in the skin and mucous membrane of the nose. The site of commonest occurrence is the mucous membrane over the anterior and lower part of the cartilaginous septum (locus Kiesselbachii). Not infrequently the tortuous vessels encroach on the floor of the nose, where they are difficult to see.

If these naevi are more prominent, and of a more tumour-like appearance, they are called angeiomata. Both forms are congenital, but may also be acquired; in the latter case from passive hyperaemia in the nasal cavity due to general or local diseases; for instance, from heart disease, plethora, constipation, etc. If they are seated on the septum nasi, they are a frequent cause of nose-bleeding.

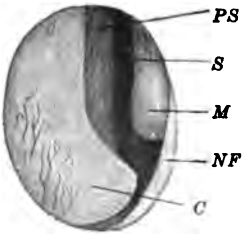


Fig. 30.—Dilatation of the vessels on the projecting septum (crista septi). *PS*, foreshortened part of the septum; *S*, meatus (narrowed); *M*, lower concha; *NF*, edge of the wing; *C*, crista septi.

Treatment.—The treatment of these naevi on the external nose is done by repeated scarification with a small knife, to the effect that the dilated vessels become subsequently obliterated. I use for this purpose a needle commonly used for paracentesis. Larger vessels must be incised; *Lassar* uses an electric scarificator. In cases of flat naevi where the redness is even, the galvanic current is useful. The electrodes are put on and a current of 2 to 3 *ma.* sent through. The duration of the sitting, which must be repeated twice or thrice a week, lasts from ten to twenty minutes. Matters can be helped by washing the parts alternately with hot and icy cold water. Drugs are of no avail. Under all circumstances the underlying malady must be treated. (For the treatment of the naevi of the nasal septum see p. 66.)

Acne Rosacea ("Blotchy Face"; Angeiectatic Erythema).—Acne rosacea attacks almost only the skin of the nose, more rarely the neighbouring parts of the face. It is merely a redness having become permanent and stationary through the angeiectatic dilatation of the blood-vessels, and is almost always accompanied by acne and complicated by hyperplastic growth of connective tissue, which gives rise to the affection known as rhinophyma (bulbous nose). In this stage the nose is very much thickened and enlarged, but to the touch always soft. It might be very exuberant and covered with nodes and lumps, and of a blue-red color; the dilated vessels are often found of the size of a knitting-needle and very prominent. In this way, extraordinary disfigurement may be produced.

Etiology.—The most common cause is alcoholic excess; but teetotalers may also suffer from it. Further, chronic indigestion, catarrh of the stomach or intestine, frost-bite, and in women diseases of the sexual organs, disturbances of periods, and uterine diseases may play a great part in exciting it. Possibly there is also some congenital predisposition, which *Lassar* sought in the uncommonly capacious size of the ducts of the sebaceous glands. The bulbous nose is met with mostly in men.

Diagnosis.—The diagnosis is not difficult. Localization distinguishes acne rosacea from acne vulgaris, which latter is seldom limited to the nose. Confusion with syphilitic disease or hypertrophic lupus should not occur, for acne rosacea never becomes ulcerated. The bulbous nose, if it is very large, might be confounded sometimes with rhinoscleroma, but can always be distinguished from it by its softness, whereas rhinoscleroma is as hard as ivory. On pressure with the fingers comedones might be expressed from the former.

Prognosis.—The prognosis is not unfavourable, if only the treatment is continued for a long time, and if it succeeds in removing the exciting causes.

Treatment.—The treatment aims at counteracting the causes. In the milder cases, especially if acne is a prominent feature, the sulphurous or resorcinous ointments can be resorted to, as in acne vulgaris. (See p. 51.) Ectatic vessels have to be scarified or incised. The rhinophyma can be successfully removed with the knife, under local anaesthesia (basal decortication). The left index-finger is pushed up into the nostril to expand the skin; and a normal nose, so to say, can be peeled out. Then under a cover of an iodoform dressing, recovery soon takes place, and a new skin is formed from the epithelial cells remaining in the glands.

II. MALFORMATIONS AND DEFORMATIONS.

Malformations of the nose, such as absence of the whole organ or parts of it, double nose, and congenital fissures, occur so seldom that we can here omit a discussion of these anomalies. Of practical importance are: (1) Adhesions (synechia and atresia nasi); (2) deformations of the septum nasi.

ADHESIONS OF THE NOSE.

They represent membranous or osseous, rarely cartilaginous, connections between opposing parts of the nasal cavity, and are known as *synechiae*, if the connection is formed by a band-like or string-like connective tissue; and as atresia, if they block more or less completely the nasal cavity like a separating wall or diaphragm.

Synechiae.—Adhesions are found in various parts of the nasal cavity; the most common is that between the lower concha and the septum. Rarely congenital, they are produced oftener through ulceration from syphilis, diphtheria, lupus, and still oftener, they are met with after cauterization and operations, if the two opposing parts of mucous membrane come in contact with each other after they have been deprived of their epithelium. At first the two surfaces stick together, and by organization of the connecting tissue, a permanent adhesion is formed. Small synechiae produce little or no trouble. More extensive adhesions may impede the respiration, and give rise to various disturbances, as headache, asthma, sneezing, coughing paroxysms, etc.

Diagnosis.—The diagnosis can be made by anterior rhinoscopy, eventually by the probe, and with the aid of cocaine and adrenalin.

Treatment.—Small adhesions, which do not cause any trouble, are better let alone. Larger ones, however, have to be divided with nasal scissors (see Fig. 40), a small knife, or galvano-cautery. Osseous synechiae should be severed by hammer and chisel. We must not be satisfied with the simple separation, but endeavour to cut off a portion of the adhesions in order to prevent a re-adherence.

For the after-treatment, it is sufficient to prescribe a menthol preparation or the daily application of renoform.

℞. Mentholi.................................0.3–0.5
Chloroformii.............................5.0
Det. in vitro flavo.
S.—Three drops several times daily to be poured on the palm and sniffed up.

Eventual re-adhesions must be separated by the probe. In certain cases it is necessary to put between the two cut ends, by means of a dressing forceps, a plate of tin-foil or suitably cut piece of cardboard, of course properly disinfected, and changed every second day.

Atresiae.—Occlusion may occur at both anterior and posterior openings of the nose. Occlusion of the anterior opening, though uncommon, might happen congenitally or through syphilis, lupus, after burns, and after cauterization. Treatment consists in the dissolution of the occlusion by knife or chisel, and afterwards in keeping apart the cut surfaces.

The occlusion also of the posterior opening, atresia of the choanae, does not often occur. Atresia may be unilateral or bilateral; complete or incomplete. It is mostly congenital and osseous, but also acquired, especially through syphilis, rhinoscleroma, or injury. With unilateral osseous occlusion may very often be noticed asymmetry of the palate, face, and even the whole half of the body. *Zarniko* thinks that the congenital nature of the choanal occlusions is not proved, and sees mere coincidences in the said anomalies.

Symptoms.—In cases where both choanae are occluded, respiration through the nose is prevented; the patient cannot sneeze, the sense of smell is lost, and taste imperfect. Speech has a nasal sound (rhinolalia clausa). All these symptoms are much less pronounced if the occlusion is incomplete or only unilateral. Sometimes the patients learn how to overcome these defects.

Diagnosis.—The diagnosis can be made by anterior rhinoscopy, if the nose be roomy enough, with aid of cocaine; better results, however, may be obtained by posterior rhinoscopy and digital examination of the naso-pharynx. The probe will decide the nature of the occlusion, if osseous or fibrous. The following points must be considered: the imperviousness of one or both choanae to air and water, the loss of smell and taste, the difficulty or disability of sneezing.

Treatment.—This can only be of an operative nature. A membranous occlusion can be cut through by knife or galvano-cautery; an osseous occlusion by hammer and chisel. The opening must be enlarged and the separated surfaces kept apart. It is often necessary, in order to make the place of operation more accessible, to widen the aditus by partial resection of the turbinate bones. Re-adhesions and re-narrowing of the operated parts may be prevented by the insertion of small india-rubber tubes or swabs, which must be changed every second day. The difficulties of the after-treatment often frustrate the success of the operation, and may render a second operation necessary. It is therefore better, especially if the discomfort complained of by the patient is only slight, not to operate at all.

DEFORMATION OF THE SEPTUM.

Alteration in the form of the septum nasi may be classified as: (*a*) simple deviations, where the septum deviates from the middle line in such a way that the convexity on the one side corresponds with a concavity on the other. (*b*) Protuberances, or partial thickenings (cristae et spinae) if the septum is thickened by osseous or cartilaginous excrescences. (*c*) Combinations of both deformities, where the protruding part shows also thickening in addition.

Etiology.—According to *Schech*, there is scarcely any person without a deviation of the septum. This is probably an exaggeration; in any case, however, deviations are very frequent; I found 71 per cent.; and, as *Zuckerkandl* has shown, Europeans are more often affected than non-Europeans; so also are males more often than females. Various explanations of its origin have been brought forward. In most cases it is due to an unequal growth of the nasal skeleton: the septum, growing more quickly than the other bones, and being fixed above and below, has to deviate to one or either side. The cause of this unequal growth is not known, but the acceptation of such a physiological deviation serves to explain why deviations of the septum are relatively seldom found in children. The jaws, indeed, grow quicker from the beginning of the second dentition, *i. e.*, from the seventh year of life. A second cause may be found in injuries, such as a blow, thrust, or fall upon the nose. In severe accidental deviations of the cartilaginous septum a dislocation of the membranous portion of the septum from the cartilaginous portion sometimes occurs (dislocatio septi nasi). Finally, a deviation may be brought about, in some cases, through the effect of pressure by either physiologically or pathologically hypertrophied turbinals.

Prominences and protuberances of the septum are mostly of traumatic origin; but they may also originate, as do simple deviations, in unequal growth.

Anatomy.—Deviation occurs mostly in the cartilaginous portion; frequently, the septum protrudes towards both sides, like an **S**, or it is shaped like a terrace and gradually slopes; in other cases it shows ridges or serrations, and sometimes it is sharply bent, forming an angle. The prominences or protuberances are of a spine-like or conical shape (spina septi), or they run as a crest or ridge, corresponding to the upper margin of the os vomer, from below and in front, upwards and backwards (crista septi).

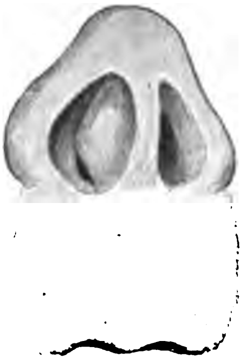


Fig. 31.—Deviation in the lower septum towards the right. Dislocatio septi nasi.

Symptoms.—Small deformities are usually without any symptoms and are discovered accidentally. Great thickening and excrescences often cause much difficulty in nasal respiration. The patient is unable to blow through the narrowed side, has to retract the secretions into the throat, has frequent resort to the use of the handkerchief, and speech becomes more or less nasal. The sense of smell is diminished, and in time, hearing is completely lost. Many nervous reflex symptoms complete the picture. Very prominent deviations or protrusions may cover the opposing turbinal, displace or flatten or even become continuous with it (Fig. 31). The nose itself is often oblique, especially if the deformity is in the lower or anterior part. On the other hand, the dorsum of the nose may form a straight line, though the deviation may be extreme.

Diagnosis.—Frontal inspection and probing are mostly sufficient to recognize the deviation. Greater is the difficulty, at least for the inexperienced, if the deviation is seated near to the entrance, when by its red colour it gives the impression of a tumour. In such a case the inspection of the nasal cavity is also made difficult, if not impossible. Otherwise a confusion of deviations with any kind of tumour or polypus of the mucous membrane is hardly possible, as both are of a much softer consistency. It is also possible, by carefully sounding, having previously applied cocaine-adrenalin, to decide what is septum and what belongs to the lateral wall.

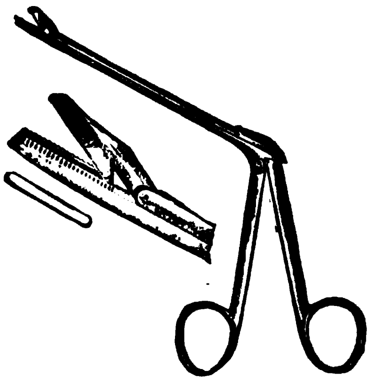


Fig. 32.—Cutting forceps (*after Grünwald-Struycken*).

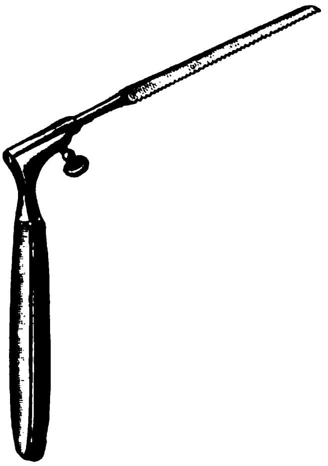


Fig. 33.—Nasal saw (*Schötz*).

Treatment.—Treatment of the excrescences and deviations is frequently very troublesome and unsatisfactory; and therefore we recommend operative procedure only in stringent cases, viz., where the complaints of the patient are urgent. The crests and spines, if they are cartilaginous, are removed with a probe-pointed knife, or with *Grünwald's* cutting forceps (Fig. 32), or with the saw, spoke-shave, chisel, or double chisel of *Schötz*, *Krause*, *Cordes* (Figs. 33, 34). The saw must be used from below and directed upwards.

In the after-treatment synechiae or re-adhesion must be prevented. This is a tedious business. Plugging of the nose (p. 66) is only necessary in severe haemorrhage. The iodoform or xeroform plugging should remain from one to three days. If the operation was done under infiltration anaesthesia, after *Schleich*, it is always well to plug, on account of the expected reactionary after-bleeding. Wherever it is possible, plugging should not be done, for it is not only very troublesome in the doing, but often found more inconvenient to the patient than the operation itself, and is also not without danger on account of retention of the secretions, infection of the middle ear, and retardation of the process of healing.

The treatment of the deviation may be done by operative or orthopaedic methods.

Orthopaedic treatment, which should be tried in cartilaginous deviations in youthful patients, has not been found a lasting success. Children can never tolerate, for any length of time, the stiff tubes or ivory rods which are inserted into the narrowed passage. Neither can I recommend the use of the pressure forceps, designed by *Jurasz* for the purpose of fixing the septum in a straight line. *M. Schmidt* makes the statement that he saw necrosis follow its application. I think it much better to postpone the treatment of deviations in children to a later age, if the case is not urgent.

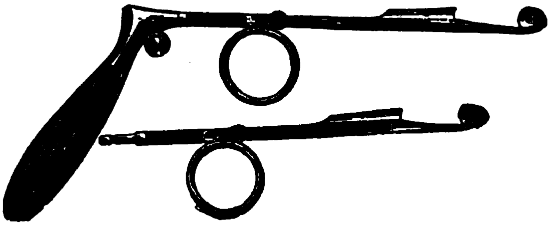


Fig. 34.—Double chisel.

Operative Methods.—1. *Method of infraction:* The deviation is divided by a particular method of cutting; it is then straightened and kept in the right position by stiff tubes or a tampon. I use successfully *Asch's* cutting forceps. This consists of a smaller concave and a broader convex blade. The hollowed smaller blade is inserted into the narrowed, the convex one into the wider, nostril. The bone is cut crosswise, put straight with a pressure forceps, and then kept in position by tubes of vulcanite such as are used for drainage. In the very prominent and low-seated deviations, it is often very difficult, not to say impossible, to introduce the small blade over the convexity of the septum. *Réthi* uses for the cross-cutting a small blunt-pointed knife.

Resection: This was recommended by *Hartmann* and *Petersen,* improved later by *Krieg;* and modified by *Cholewa, Bönninghaus, Killian, Zarniko,* and others. The mucous membrane of the narrowed side is cut curvilinearly or angularly, the base of the angle being directed backwards, and the mucous membrane is then pushed aside, together with the perichondrium. Then the cartilage is carefully perforated with a galvano-cautery or gouge, so as not to damage the mucous membrane of the other side, which by means of a raspatory or elevator, is likewise detached, together with the perichondrium. Then the piece of cartilage thus freed and laid bare is removed with a *Heymann's* sharp cupped-forceps (Fig. 35). A small perforation of the mucous membrane of the other (concave) side is of no significance. There is usually free hemorrhage, and to stop it, a sufficient number of swabs should have been prepared. The method is not easy, but has the advantage of not requiring any special after-treatment.

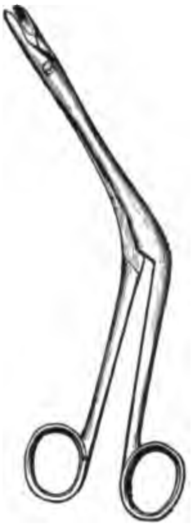


Fig. 35.—*Heymann's* sharp cupped forceps.

Injury to the Nose.—In spite of its prominent position, in the very exact sense of the word, the nose relatively does not very often suffer injury. Contusions or open wounds are easily recognizable and treated on general lines. Fractures of the nasal bones are divided by *Bergmann* into transverse and comminuted fractures. In the former, displacement is often absent, and can then be treated conservatively; if there be displacement of the fractured parts, they should be replaced, and kept right by fixing the bones, by means of plugs within the nose. Comminuted fractures require operation.

Great force usually injures the septum, mostly the cartilaginous part of it. Haematoma, deviations, dislocations, and fractures of the septi nasi have been observed. The haematoma, on account of an infective focus in the mucous membrane, nearly always leads to:

Abscess of the Septum.—Some authors have occasionally observed a purulent perichondritis as a cause of—not traumatic—septal abscess. One finds the dorsum and the entrance of the nose infiltrated, swollen, red, and tender; the redness may also spread over the neighbouring skin of the face. The anterior part of the septum bulges forwards, mostly on both sides; the swelling, being tense and elastic, blocks more or less the additus ad nasum. If pressure applied to the one side causes the other side to bulge more, it is a sign that the cartilage has been perforated. In acute cases fever may be present; but not in the more subacute or chronic cases.

Treatment.—Treatment consists in freely opening the abscess and disinfecting and afterwards tamponading. Haematomas should also be incised, as otherwise infection can barely be avoided.

Deviations, Dislocations, and Fractures of the Septum.—These are not always easy to recognize on account of the accompanying swelling of the parts. It is not common for these cases to come under observation soon after the accident; usually, only the after-effects—the permanent deformities—are seen; and it is only possible from the history to infer that there has been an accident, and because the nose bled, etc., that the deformity originated in an injury.

Treatment.—Treatment aims, as in other fractures and dislocations, at the replacement of the parts and at keeping them in the right position, by means of plugging the nose. To put the fractured parts straight, closed pressure-forceps, a probe, or catheter may be used.

III. HAEMORRHAGE.

Bleeding from the nose, having regard to the exposed situation of the organ and great vascularity of the mucous membrane, is very common. Generally, young persons are more subject to it than their elders. We may divide haemorrhages into:

A. Haemorrhages into or under the mucous membrane (ecchymosis, haematoma, etc.).

B. Free haemorrhage.

A. BLEEDING INTO OR UNDER THE MUCOUS MEMBRANE.

Ecchymoses do not often occur in the nose. Their presence points to a general disease of the circulatory organs. Of more importance practically are haematomas, which are usually due to the traumatisms before mentioned. They appear as dusky, red, or even black, tumours of a smooth, tense, and elastic surface, on both sides of the septum. They may be so large that the entrance of the nose may become entirely blocked. As to their treatment and their leading to suppuration, see above.

B. FREE HAEMORRHAGE.

This is produced either by local or general causes. *Local causes* are: injuries, through impact, thrust, fall on the nose, nose-picking, operative manipulations, foreign bodies or catarrh of the mucous membrane, ulcers, naevi, vascular tumours, such as polypi, sarcomata, etc., and also by fractures of the base of the skull.

General causes are: disease of the circulatory organs, heart, lung; nephritis, especially chronic granular kidney, cirrhosis of the liver, arterio-sclerosis; diseases of the blood, such as haemophilia, scurvy, chlorosis, pernicious anaemia, leukaemia, psuedoleukaemia; further, poisoning from phosphorus, lead, etc.; acute infections, for instance, influenza, scarlatina, measles, whooping-cough, typhoid, typhus, smallpox, septicaemia, etc.

Bleeding in these cases is due to high arterial pressure or passive hyperaemia or disease of the walls of the blood-vessels. The nasal mucous membrane seems to be the seat of predilection. A sudden lowering of the outside atmospheric pressure may cause bleeding from the nose, as is observed sometimes in mountain-climbers, aëronauts, and caisson-labourers.

Not infrequently nose-bleeding takes place in connection with abnormal processes in the sexual organs. It may occur during disturbances of the menses, and sometimes replaces them altogether (vicarious menstruation); it may disappear, however, if pregnancy intervenes. It here perhaps relieves reflex hyperaemia of the nasal mucous membrane, as in some forms of sexual excesses. At times nose-bleeding may precede menstruation.

A curious case observed by *Wunsch* may be quoted here, in which slight bleeding of the normal tonsil immediately preceded each menstruation.

By far the most common cause of nose-bleeding is *an injury to ectatic, dilated blood-vessels* of the cartilaginous septum. (See p. 53.) These naevi correspond to those vessels described by *Kiesselbach* as occurring at the lower anterior part of the septum, and which appear to form a kind of cavernous tissue, as in the lower turbinate body, and are also found sometimes on the floor of the nose. These vessels are so numerous and seated so superficially that a relatively slight injury is sufficient to cause them to bleed, as, for instance, through coughing, sneezing, stooping, etc. Tight collars also cause hyperaemia by pressure on the blood-vessels of the neck. Young people are often very much pestered by habitual nose-bleeding. According to *Donogány*, this is due to atrophy of the nasal mucous membrane at the locus Kiesselbachii (rhinitis sicca anterior), produced by repeated catarrhs, and haemorrhages very frequently occur just in this spot, as the result of customary picking of the nose.

Symptoms.—*The nose bleeds in single drops*, whence the name epistaxis is derived; or the bleeding is in the nature of a more continuous flow. It may stop after a while, and sometimes recur without visible cause, especially if the patient is careless. If the bleeding is profuse, much of the blood flows backwards into the throat and the patient then swallows it or spits it out, or it finds its way out through the other nostril by way of the choanae. It may happen to reach the larynx, when it will cause irritation and paroxysmal coughing, and is then expectorated. In some cases epistaxis is quite sudden; in others, especially in plethoric persons, it is preceded by symptoms of congestion or hyperaemia, headache, dullness, vertigo, and tinnitus; here the bleeding has a good effect, like a venesection or tapping. Severe haemorrhage may be followed by acute anaemia, fainting, and even death has been recorded.

Diagnosis.—We should always endeavour to discover the source of the bleeding. This will be mostly found to be some torn vessels at the locus Kiesselbachii. In order to see it, the speculum must be introduced with the blades directed upwards and downwards. If we do not see the patient until some time after the bleeding, we will have no difficulty in recognizing the dilated vessels of the septum. Nevertheless, such vessels are also found in healthy noses. After the septum, the floor of the nose must also be inspected. If here again nothing is detected, the entire nasal cavity must be examined. Occasionally some scabs are found, which will indicate the seat of the haemorrhage.

If a patient has to be examined while he is bleeding, the blood should be carefully sponged or dabbed up without wiping or rubbing, or we may plug the spot for a short while. It is always well to clean the cartilaginous septum before we make our examination; but with all our gentleness in handling, we must be quick, and having again removed the tampon, we must make a diagnosis, so to speak, at a glance.

If we fail to discover any source of bleeding within the nose, we must endeavour to find out if the blood does not come from the throat, stomach, or lung. Not infrequently, as has been already mentioned, the oozing blood flows backwards down into the throat, especially if the patient reclines his head, and then from the throat it may reach the lung, or it may be swallowed and brought up by coughing or vomiting. For this reason we should always inspect the nose in cases of doubtful haematemesis or haemoptysis.

Prognosis.—Nose-bleeding should not be lightly regarded, especially in anaemic or weak persons. Prognosis is based, of course, on the cause of the bleeding. It is almost always favourable, should the bleeding be from the cartilaginous septum, and in this case it can be easily treated. It is more uncertain, or even unfavourable, in cases of general disease, like haemophilia, arterio-sclerosis, or cirrhosis of the liver. It is an ominous sign in fracture of the base of the skull.

Treatment.—In plethoric patients, or if substituting menstruation, nose-bleeding has a beneficial effect and needs no local treatment, if it is not very severe or does not last too long. Under these circumstances a hot foot-bath and a dose of an aperient mineral water are all that is necessary. It is well to add to the hot water for the foot-bath one or two handfuls of common salt or mustard. As it is mostly the anterior part of the septum which bleeds, a piece of cotton-wool may be introduced into the respective nostril, and then the nose can be compressed with the fingers. This will soon stop the bleeding. Cotton-wool soaked in *chloride of iron* is not to be recommended, though sometimes useful, for it has an undeniably irritant effect. To hasten the cessation of the bleeding, one may use *ferropyrin* instead of chloride of iron, which is cleaner and not an irritant; or *renoform wool* may be applied with advantage.

Cold applications, sniffing up ice water, or astringents are of little avail, if, indeed, they do not serve to aggravate the trouble. It is often best to seat the patient quietly, with the head erect, or lightly bent forwards, avoiding entirely sneezing or blowing the nose, or wiping, and then to change the plug only if it is fully saturated with blood. The changing of the tampon must be done very gently and without haste; the same advice applies to doctor and patient alike. If the bleeding spot has in some way or other been discovered, a small pledget of cotton-wool steeped in adrenalin first, and then another in cocaine, should be pressed against the part. The site is then cauterized with *chromic acid* or *trichloracetic acid*, or with the flat-pointed galvano-cautery. (See Fig. 28.) After cauterization the patient frequently sneezes, but usually this does no harm, for the eschar is sufficiently secure. In slight haemorrhages, simple insufflations of renoform should be sufficient. The powder mixes with the oozing blood and forms a sort of coagulum, and it has, besides, a styptic effect, on account of its containing adrenalin.

Post-operative haemorrhage often ceases of its own accord if the patient only keeps quiet; otherwise the nose must be tamponaded. This can be performed through the anterior or posterior nares. Tamponading, however, which is not entirely harmless, and is certainly troublesome, should not be resorted to except in cases where one cannot find the bleeding point, or if bleeding is so severe that one has no time to spare and other measures have failed. Luckily, however, this happens but seldom. For plugging through the anterior meatus one introduces with a dressing forceps small strips of iodoform or xeroform gauze. The former is, however, apt to irritate and to cause sneezing. It is often quite sufficient to press a simple tampon against the bleeding point.

If bleeding continues, the whole nose must be filled with gauze, and care must be taken that the gauze does not fall down behind into the postnasal cavity. The tampon must be removed after two days, and if it offers much resistance, only a part of the gauze must be withdrawn and cut off. The rest can remain in situ for another day or so, until it becomes loose and slippery on account of the nasal secretions. *Lublinski* introduces, in severe cases of bleeding, a folded strip, 2 cm. broad, of dermatol gauze into the nose. The ends of this strip are left depending from the nostrils, while the folded portion lies in the depths of the nasal cavity, which is then also entirely filled with aseptic wool.

Plugging from behind should be done only in case of urgency, *i. e.*, where anterior tamponade has failed.

It is customary to use for posterior tamponading *Bellocq's* cannula sound. Unfortunately, this frequently fails in an emergency, and is not always slender enough to pass a narrow or irregular meatus. It is, therefore, better to use a long elastic catheter, which is pushed through the lower meatus into the postnasal cavity, until its end becomes visible behind the soft palate; it is then seized with forceps and pulled forward so far that a tampon of aseptic cotton-wool, enveloped in iodoform gauze, can be tied to it. The tampon should be at least half the length of the patient's thumb, and before it is used, it must be tied in the middle of a strong silk thread about 60 cm. long. The next step now is to attach one end of the thread to the catheter by passing it into the eye of the catheter and tying it firmly. The catheter is then slowly drawn back through the nose, and in this way the tampon is pulled into the choanae. As a rule, it will be well to aid this procedure by the finger from behind, in order to pilot the tampon into its place. The catheter is now withdrawn, and the nasal end of the thread is held taut, whilst and until the whole nasal cavity is packed from in front. Finally, the nasal and oral ends of the thread are knotted, and the loop placed behind the patient's ear. If one is in possession of waxed silk thread, this can be pushed through the whole length of the catheter, until it appears at its eye; it can then be seized and tied about 30 cm. from its end round a tampon; and the rest of this proceeding is then the same as in the previous one. The oral end of the silk thread is very apt to cause choking. It is, therefore, better to sometimes tie

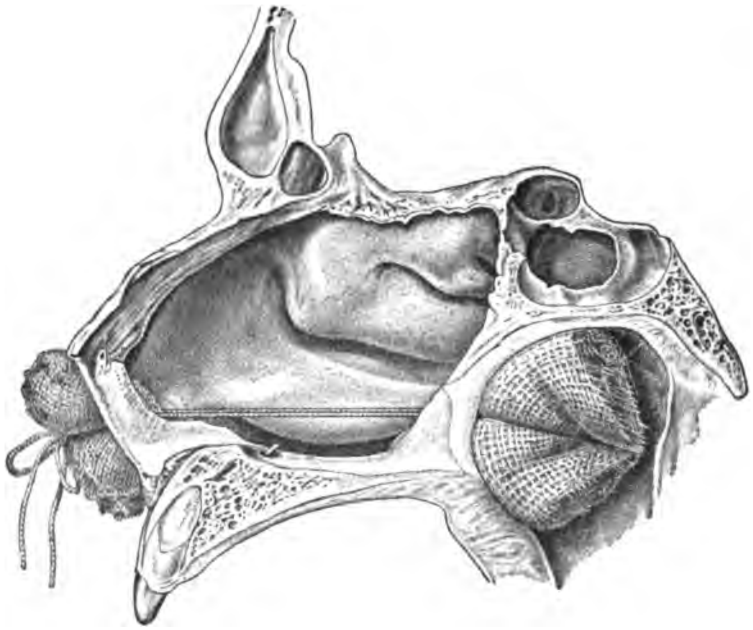


Fig. 36.—Position of the tampon (*after Hochenegg*).

both ends of the thread to the catheter and to guide both of them through the nose. *Hochenegg*, in such a case, in order to firmly fix the choanal tampon, ties the two ends over a pad of cotton-wool placed in front of the nostril. The tampons should not remain in situ longer than forty-eight hours, otherwise they are liable to give rise to inflammation of the middle ear, or even meningitis, through retention of the nasal secretion. If necessary, tamponade may be repeated. (See Fig. 36.)

In dangerous bleeding injections of gelatin may be tried;

20 to 30 c.c. of warm fluid gelatin are to be injected into the nose. It here sets quickly under the cool current of air.

Internally, extractum secalis cornuti or ergotin (8 : 200 of water), of which one tablespoonful every two hours may be given; or hydrastis canadensis, 25 drops three times daily; or stypticin, 3–5–8 tabloids a day, may be given.

Any possible underlying systemic disease should be borne in mind and treated according to medical indications.

IV. RHINITIS ACUTA (CORYZA; "SNUFFLES"; "COLD IN THE HEAD").

The acute inflammatory processes of the nasal mucous membrane offer, in spite of their common occurrence, no little difficulty as regards their terminology. None of their classifications, *sensu strictiori,* is exact, neither clinically nor etiologically. For practical purposes it is perhaps best to regard them from the etiological standpoint, if this is admissible.

Etiology.—Acute rhinitis arises from many causes. In the imagination of the public, and often also of the physician, cold plays the chief rôle. The so-called "cold in the head" (coryza, rhinitis catarrhalis acuta) is supposed to be always a consequence of "catching cold," though it would often be difficult to prove it. What generally is called "snuffles" is probably always an infectious disease, due to micro-organisms. The "cold" provides only the predisposition, preparing, through alteration of the circulation, the soil for the invading bacteria; as we likewise see it in other infectious diseases, though we cannot always prove it by experiment. In simple coryza artificial transmission—if we except the one case published by *Beckmann* (1902)—has not been successfully established; neither has a special microbe been found; probably because the disease called coryza is caused by many and various organisms. In any case, that many members of one family, one after the other, often become ill, the concomitant disturbance of general health, together with enlargement of the spleen (noticed by *Friedrich*), all seem to point to the infectious nature of rhinitis acuta.

Though coryza may, in a certain sense, be a disease of cold, like angina, it often prevails in the warmer seasons of the year, especially as, during continuously dry weather, dust, and with it bacteria, are very much increased.

During warmer weather we also take less care of ourselves, and expose ourselves more to "refreshment," which often means sudden change of temperâture. This would help to explain the common occurrence of the " cold " in spring and summer. It is easy to see that children become affected oftener than adults. Persons who have once suffered from "colds in their head" are, so to speak, predisposed, and contract it, sometimes quite regularly, in the transitory seasons.

There can be no denying the infectious nature of those colds, which appear as symptoms or complications of other infectious diseases (scarlet fever, measles, smallpox, influenza, etc.).

Chemical or mechanical irritations may cause coryza. Of chemicals are: especially iodine, in its various medicinal forms, and the vapours of various gases or acids, which produce it, without causing general symptoms. Of purely mechanical action are soot and dust ("railway cold") and many other substances of organic or inorganic nature. Frequently both actions are combined, as, for instance, in certain trades and professions. Thus we often hear of "coryza professionalis." *Roepke* records not less than 150 various trades or professions where those practising them may get an acute catarrh of the upper air-passages, leading sometimes to profound alteration of the tissues (ulceration, perforation of the septum, etc.). Plugging, cauterization, and other operations may also be responsible for acute catarrh; even a severe blow upon the nose may give rise to an attack of coryza, most probably by reflex action. (See p. 126.)

That form of coryza which occurs through the aspiration of pollen dust, and known as "hay-fever," as also the "cold" frequently observed in neurasthenic or hysterical persons, and passing under the name of "nervous coryza," will be further considered below.

Symptoms and Course.—Acute rhinitis is often preceded by general symptoms, as general malaise, giddiness, shooting pain, headache, or even an increase of temperature. At the same time with these, or following them, the patient complains of a burning sensation or irritation in the throat, of dryness in the nose, or rather at the back of the nose, and sneezes frequently. The discharge at first is watery and thin, and on account of its containing *ammonia*, is acrid and irritating. After one day or so the secretions become opaque, more viscous or mucopurulent, and of a more yellowish-green colour; finally the quantity again decreases until it ceases entirely. While the catarrh is at its height, taste and smell are diminished or even lost. Respiration through the nose, in consequence of the swelling of the mucous membrane, is impeded or even impossible; and the voice becomes nasal (rhinolalia clausa) on account of the obstruction. This frequently occurs on both sides; but just as often one side only is affected, and it may then "jump" over to the other side. It is curious that in open air the nose feels freer than in a close room, where again it soon becomes blocked.

Coryza lasts only a few days, often not more than twenty-four hours, but may also persist for some weeks, especially if the catarrh spreads as well to the neighbouring mucous membranes. Complications are sore throat, fissures, and pustules at the entrance of the nose or eczema on the lips ("chapped" lips), lachrymation, conjunctivitis, tonsillitis, disease of the middle ear, of the frontal and ethmoidal sinuses and antrum, catarrh of the upper air-passages, bronchitis, laryngitis, etc.

Pathology.—The nasal mucous membrane is swollen and red, sometimes ulcerated; and the vessels are tortuous and dilated. The nasal passages are full of secretion, which, according to the stage of the disease, shows differences of consistency and colour. Microscopically there is at first hyperaemia and round-celled infiltration. Subsequently, one finds the tissue oedematous and showing loss of surface epithelium. The discharges in the beginning show a large quantity of epithelial cells and lymphocytes; later on, the mucous cells therein are more numerous, and in time there may even be an admixture of blood.

Diagnosis.—Acute rhinitis is, after all, easy to recognize. Difficulties are only possible at the beginning as regards the etiology. The decision whether there is present only an ordinary cold or a nervous form of the disease; whether the rhinitis only means the onset of an infectious disease, as in measles in children, can only be made after a certain lapse of time. Nervous rhinitis is characterized by the shortness of the attack, which comes like a flash and disappears as quickly, together with the presence of other nervous symptoms.

In infants, long-standing snuffling should make us suspicious of hereditary syphilis; and in older children, of adenoids. If there is frontal headache, with a feeling of tension and pressure at the root of the nose, complications in the region of the frontal sinus are very likely, and difficulties of hearing or noises in the head point to affection of the middle ear. Otitis, on the other hand, manifests itself nearly always by the sudden onset of pain behind the ear, often at night. Investigation will tell us that the patient has sneezed or blown his nose violently, or has sniffed up some solution. In children the ear is much oftener affected than in adults, for the reason that the Eustachian tube in children is relatively shorter and wider.

Prognosis.—The prognosis is mostly favourable. A transition from the acute to the chronic form often occurs. It is less favorable if neighbouring organs become affected, but here also recovery, after a time, is mostly complete. In infancy acute rhinitis always means a serious disease; the same may be said of old people who are liable to broncho-pneumonia, etc.

Treatment.—In the treatment of acute catarrh, we should always remember that a cure can only be effected by the natural forces, and that we have, therefore, to leave matters to the resources of the organism. The treatment essentially of acute rhinitis, then, is only directed in a hygienic and dietetic way, viz., by removing or abstaining from all nuisances or irritants which could interfere with the reëstablishment of healthy conditions. It is not necessary to keep to the room or bed if there is no fever or other complication. In mild attacks, going out of doors should always be allowed, for the patient is much better in the open air than in a closed room. We must not, of course, expect too much by this method, yet local treatment will not accomplish more by itself. In most cases a "cold" recovers by itself without any treatment, or in spite of it; therefore, it is better to abstain from being too officious. An early hot bath might, perhaps, cut short the cold. I have often seen benefit accrue from phenacetine (1 gramme doses every two or three hours), whereas the much-praised opium has been of no use. Recently *Bier's* treatment, as I have observed in my own case, and in that of other patients, has shown itself very efficacious. An elastic band or india-rubber tube is passed round the neck just so tightly that the circulation in the deeper veins and arteries should not be interfered with. This means a pressure of about 25 to 30 mm., and the band remains round the neck for from two to five hours. A handkerchief instead, however, might be employed. Through this bandaging, a hyperaemia of the head is produced, which very soon shows itself by the flushing of the face and conspicuous swelling of the veins. Another method which is supposed to have an abortive result, but is difficult to carry out, consists in eliminating all fluids of whatever kind from the diet for two days. In very troublesome patients only a tablespoonful of tea or milk or a wineglassful of water should be allowed.

The treatment should mostly be directed to relieve the trouble. The most unpleasant symptom, the obstruction of the nose, can be relieved, at least temporarily, by sniffing a powder composed as follows:

℞.	Menthol		0.1–0.2
	Pulv. cubeb.		
	Acid. boric.	āā	0.5
	Pulv. talci		10.0

SIG.—Sniff up a pinch through both nostrils.

Very useful is a combination of menthol and cocaine, such as:

℞.	Cocain. hydrochlor.		
	Menthol	āā	0.1–0.2
	Sacch. lact	ad	10.0

One can also recommend renoform powder. If the patient, owing to the swelling of the mucous membrane, has a difficulty in sniffing up the powder, it is advisable to administer inhalations of menthol. (See p. 56.) Sometimes painting the parts with adrenalin 1 : 1000 or cocaine 5 to 10 per cent. affords momentary relief. The use of cocaine, however, is not always harmless.

Formane wool, which is so often advertised and praised, as also pulverized boric acid, is somewhat more irritant. If the secretion becomes too copious, it is advisable to anoint the nostrils and upper lip with lanoline or boric vaseline, and in long-standing catarrh the application of silver nitrate will sometimes do good.

Infants who suffer from acute rhinitis are unable to suck, and must be fed by a spoon. Here sometimes instillation of liquid paraffin, 1 or 2 drops into each nostril, or small pledgets of cotton-wool steeped in adrenalin and applied three or four times a day for two or three minutes, will improve the breathing. *Naegeli* recommends instillation of a drop of cocaine-glycerin solution:

℞. Cocain	0.2
Aquæ dest.	
Glycerin	āā 5.0

Sig.—One drop in each nostril.

Tracheotomy might have to be performed in very desperate cases.

All injections, or the very popular sniffing up of fluids, or forcible "blowing," must be forbidden.

Treatment of the Complications.—Acute catarrh of the other cavities subsides *pari passu* with the acute rhinitis. Conjunctivitis might be treated by instillations of sulphate of zinc or fomentations with a solution of boric acid. In disease of the middle ear use hot fomentations over the ear, and, if necessary, perform paracentesis. In simple serous catarrh of the middle ear "Politzering" may be beneficial.

Prophylaxis.—As to prophylaxis, a reasonable hardening process has to be aimed at; workers or traders in substances which are known to irritate the mucous membrane should protest the nose by respirators or by light plugging. Rhinitis caused by iodine preparations usually subsides if the drug is discontinued.

V. RHINITIS CHRONICA.

In the chronic inflammations (chronic rhinitis, ozaena) it is still more difficult than in the acute inflammatory processes of the nasal mucous membrane to make a classification answering all requirements, as all possible diseases, whether only slightly or artificially connected with each other, go under this heading. In the strict sense of the term, chronic inflammation, only one process should be understood; that is, a chronic process characterized by swelling and abnormal exudation. For practical purposes, however, it seems advisable to distinguish between *simple chronic rhinitis,* where the swelling of the mucous membrane is more equally diffuse and moderate, and *chronic hypertrophic rhinitis* (*rhinitis hyperplastica*), where the swelling is more patchy and often very considerable. Both forms may pass from one into the other, and are the opposite of *atrophic rhinitis* (*rhinitis atrophica*), which latter, however, many authors regard as a terminal stage of the two former. This does not mean, though, that hypertrophic rhinitis invariably ends in the atrophic form. There are many cases in which both hypertrophy and atrophy may coëxist. In the following section we shall discuss together the simple chronic and hypertrophic rhinitis, which represent only different degrees of the same disease, and will treat the atrophic form in a separate chapter, as it is held to be pathologically a separate disease, *sui generis.*

1. CHRONIC RHINITIS (RHINITIS CHRONICA SIMPLEX HYPERTROPHICA).

Etiology.—Persons acquire chronic catarrh of the nose who have suffered repeatedly from acute catarrh and live under unfavorable hygienic conditions, as, for instance, in dusty or smoky atmospheres, or are exposed to irritant vapours or solid substances in minute division, etc. Narrowing of the nasal passages or deviations of the nasal septum predispose to it, because of the obstruction and disturbance of the circulation (*M. Schmidt*). Chronic rhinitis occurs in conjunction with, or as a consequence of, disease in the neighbouring cavities (empyema of the antrum, adenoid vegetations, etc.). Chronic catarrhs are encouraged by a certain catarrhal predisposition, due probably to some disturbance of the circulation, as we find it in chronic alcoholism, diseases of the heart, kidney, or liver, but also in struma and anaemia. The male sex is more prone to suffer.

Pathology.—In simple diffuse rhinitis the nasal mucous membrane is reddened and thickened in toto. In the hypertrophic form the middle and lower conchae (turbinals) are the parts most concerned. In the former the swelling is caused principally by increased hyperaemia, which, under favourable conditions (see below), tends to recovery. In the latter form there is a permanent increase of the volume of tissue; a more or less permanent swelling, due to the new formation of connective tissue. Microscopically, one finds small round-celled infiltration, especially near the surface, and a great increase of new fibrous tissue and vessels. Very remarkable is the dilatation of the cavernous spaces, the walls of which are very much thickened.

The posterior, but also the anterior, ends of the conchae, especially the lower one, may be conically enlarged, or may be pear-shaped, or they may show a smooth, warty, or indented, raspberry-like surface. These structures may be so much enlarged as to simulate a tumour, and are of a dusky red; and, if the epithelium is very thick, of a somewhat whitish colour. On the middle concha it often gives rise to the so-called *mucous polypi* (see p. 114), which many regard as tumours; but, as *Zuckerkandl* has proved, are nothing else than hypertrophic formations of the mucous membrane, *e. g.*, inflammatory products (Fig. 37).

On examination, these papillary or warty swellings are very difficult to distinguish from polypi. The broad-based swellings, which are also usually harder, are known as *polypoid hypertrophy*, in contradistinction to the more movable, stalked, jelly-like, and viscid mucous polypi. It is quite true that the latter give the impression of a new-growth, and in any case they are clinically so distinct that we are justified in discussing them later on in another paragraph. Sometimes we find besides swelling of the concha, the cartilaginous septum also; and in some very advanced cases even the bones hypertrophied.

In such cases bullous blistery enlargements may be observed, principally at the anterior ends of the conchae, but also on the septum.

Exudation is copious, seldom thin or watery, oftener mucous or muco-purulent, and then of whitish or yellowish appearance, but almost always devoid of odour.

Symptoms.—The patient complains mostly of a constant or intermittent obstruction of the nose and of "accumulation of phlegm" (mucorrhoea). These are the chief symptoms, and of any others there is little or no complaint.

The amount of obstruction depends on the degree of swelling of the mucous membrane. It sometimes "jumps" over to the other side or changes within a short time on the same side, according to the conditions of fullness in the cavernous tissues.

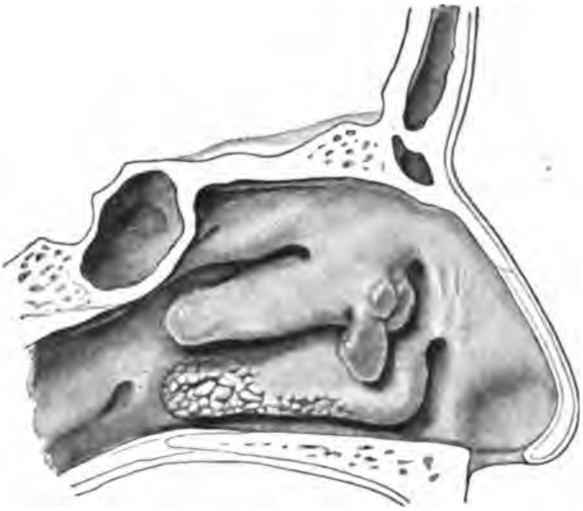


Fig. 37.—Polypi at the anterior end of middle concha. Polypoid hypertrophy at the posterior end of the middle and papillary hypertrophy of the posterior end of the lower concha (left side).

This condition again depends much on psychic influence, due to reflex neuroses (rhinitis vasomotoria, p. 125). When lying down, the side usually lain upon is the one obstructed. In marked hypertrophic rhinitis, nasal obstruction is mostly more or less constant.

Mucorrhoea is a troublesome symptom. The patient uses a great many handkerchiefs, because he has to blow his nose very often, or he complains of it only from hearsay, because he draws the nasal secretions backwards into the throat. Patients are often forced to sneeze, to clean the nose, and complain of a sensation of dryness or burning in the throat, especially in the morning, as the nasal secretions flow down into the throat during the night and remain there and become concentrated. This produces the sensation as if a foreign body were lodged in the throat. Paraesthesias, such as pricking and burning, are mostly present. In time the mucus drawn backwards or swallowed may cause unpleasant choking or vomiting. The sense of smell is in many cases diminished or lost. Other symptoms are caused by the nasal obstruction or by the spreading of the catarrh to other organs. The voice gets a nasal tone (rhinolalia clausa) and the tip of the nose reddens (p. 52). Conjunctivitis, epiphora, catarrh of the other cavities, ear disease, headache, and lowering of the general health by reason of the psychic depression are not uncommon (p. 31).

Diagnosis.—The diagnosis is made by anterior and posterior rhinoscopy. On examining from behind, the hypertrophied conchae are seen, often protruding into the naso-pharynx. We must satisfy ourselves by the probe whether the prominences are hard (osseous) or soft (swellings). Practically, it is of value to ascertain if we have only to deal with swelling due to hyperaemia or with a real hypertrophy. For this purpose we paint the swollen parts with cocaine or cocaine-adrenalin. If, after painting, the swelling subsides or diminishes conspicuously, we can be quite sure that it was due to hyperaemia and greater filling of the cavernous tissues. On the other hand, if the swelling does not subside or diminishes only a little, we know that there is real hypertrophy. Its mobility we also test by probing. It is not uncommon in nervous patients that a hyperaemic swelling subsides on the mere touch of a probe. The reflex irritability is here so much aggravated that the slightest stimuli prove amply sufficient to produce changes in the conditions of swelling (see Reflex Neuroses).

Prognosis.—Improvement, sometimes even cure, can be established by proper treatment, though the disease is obstinate and very hard to contend with. Less favourable is the prognosis in those cases of diffuse inflammation with nervous complications.

Treatment.—Treatment aims at three purposes: (1) The influence on the general health; (2) the treatment of eventual underlying disease; (3) restitution of the local condition to that of health. As regards the general treatment, we refer to what we have said on hygiene and prophylaxis elsewhere (p. 44). In this matter a suggestive or psychic treatment is of great importance. Patients suffering from chronic nasal diseases are often neurasthenics or hypochondriacs, and readily resign themselves to the small inconveniences of their sufferings if we can assure them that they have not any dangerous disease, or that there is no disposition to tuberculosis, cancer, etc.

General diseases, such as anaemia, struma, and constipation, must be treated on general lines. Adenoid vegetations and affections of the neighbouring cavities require special treatment.

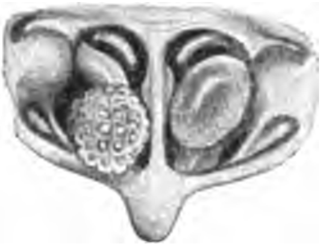


Fig. 38.—Polypoid and papillary hypertrophy of the ends of the concha in the post-rhinoscopic picture.

In the milder forms, local treatment consists in the application of astringents. I use chiefly 3 to 10 per cent. nitrate of silver, which I paint on, usually every second or third day, after locally anaesthetizing. Astringent powders I consider superfluous, because it is often not possible to apply them to the exact spot. But in order to facilitate the removal of mucus and the clearing of the nose, I order the inhalation of menthol (fluid or vapourized) (pp. 56 and 73). If this method of cleaning is insufficient, the secretions can be carefully washed away (p. 38). In cases with scanty secretions, inclined to scab formation, painting with iodine is useful. In all these procedures *the postnasal cavity must not be neglected.*

In case of more marked hypertrophy stronger remedies must be applied. The previously anaesthetized part is cauterized with lapis infernalis, chromic or trichloracetic acid, or with a galvano-cautery. If one desires to obtain a deeper effect, the pointed galvano-cautery is stabbed into several places, or slight grooves are made in the diseased part; and, in addition, into the grooves so made, one of the above-named acids can be rubbed in. By this method considerable scarring is effected. In the after-treatment synechiae or readhesions should be prevented (see pp. 55, 57), which, however, is not an easy matter.

It sometimes happens that two or three days after the cauterization, especially galvano-cauterization, symptoms of angina tonsillaris make their appearance. According to *B. Fränkel,* this is due to the invasion of infectious germs into the wound and to their spreading into the lymphatic tissue of the tonsils. *Scheller* and *Stenger* have shown micro-organisms in the tonsils after operation, which they had found before operation in the nasal cavity.

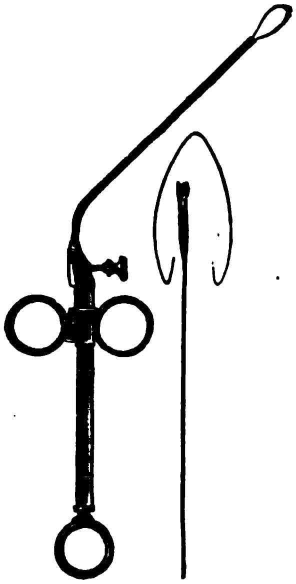


Fig. 39.—Polypus snare (*after Krause*).

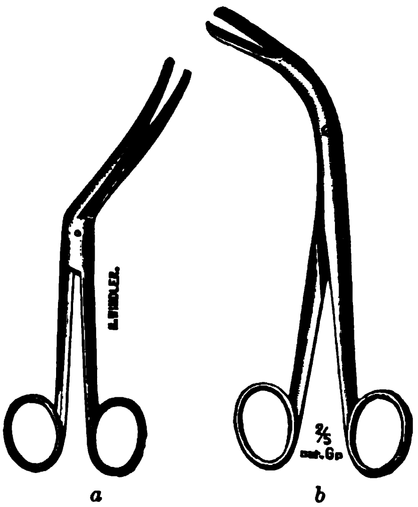


Fig. 40.—*Beckmann's* nasal scissors: *a,* For the lower, *b,* for the middle, concha.

In order to remove larger circumscribed hypertrophies from the conchae, the cold snare or, if the swelling is more broadly seated, the nasal scissors, are used. (See Figs. 39 and 40.)

Krause's snare is used in the following way: the thumb is put into the hinder, the index-finger into the upper, and the middle finger into the lower, ring; the upper and lower ring being close to the guide tube. A loop of wire just long enough to encircle the hypertrophy to be removed is then formed, and threaded through the guide tube. Under the guidance of a speculum the instrument is cautiously introduced, the loop being nearly perpendicular. It is then pushed as far as possible until the hypertrophy is reached (it is sometimes necessary to turn the handle). The hand must be steady and quick. Then the sledge is now pulled back towards the thumb ring, and thus the hypertrophy is caught and cut off.

In order to reach the posterior ends of the concha it might be necessary to remove swellings, spines, or enlargements which block the way. If we want to put the snare round the posterior end of the concha, it must be bent a little sideways. For this purpose a modification of *Krause's* snare is very useful, which is constructed in such a way that the loop is formed and bends by itself. I recommend *Jaenicke's* snare, which is introduced with the loop closed, and then the loop is opened at the desired spot. If we intend to remove a larger piece of the hypertrophied lower concha (conchotomy), a practice which is recommended in narrow deviated noses, *Beckmann's* scissors may be used with advantage. (See Fig. 40.) The instrument is introduced in such a way that the ends of the blades pass beyond the hinder end of the concha. The speculum is then carefully withdrawn; the scissors are now opened, and the end of the concha cut away. Sometimes the cutting nasal forceps of *Grünwald* (Fig. 32) are employed. In certain cases, if a part of the bone must be removed, *Hartmann's* conchotome, which is made in three different sizes, serves the purpose equally well. (See Fig. 41.)

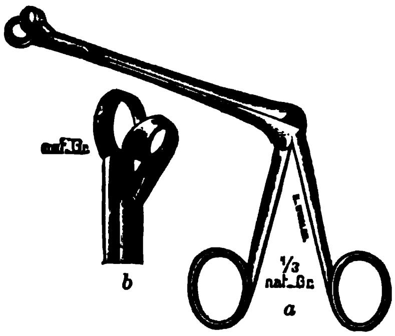


Fig. 41.—*Hartmann's* conchotome: *a*, Cutting horizontally; *b*, cutting vertically.

We strongly advise the operator not to remove too much from the concha, for otherwise the way is opened to atrophic processes, which amounts to substituting one evil for another. We also strongly condemn the operation mania of some specialists who, perhaps without proper indications, always pinch and cut about the inner nose. All this overoperating is also very apt to make the patient a neurasthenic person. It is perhaps not superfluous here, if we direct attention to the fact that all instrumental manipulations or operations should be performed according to the strict rules of antisepsis and asepsis; and that we should endeavour to perform all operations, causing as little pain as possible, by means of the application of cocaine-adrenalin. In trying to remove swellings seated far back, we should take care that the drug is not painted on too far back, for the hypertrophy may, by this means, be so much reduced that the wire loop always slips off. It is sufficient to paint in such cases the part of the mucous membrane just in front of the hypertrophy; and this has, moreover, the advantage of rendering the introduction of a speculum easier and thus the ends of the conchae can be more readily inspected. For the use of alypin in certain cases see p. 22. Plugging the nose is only necessary in rare cases, where there is severe after-bleeding.

The galvano-caustic snare has no advantages over the cold one. Its styptic effect, which is so much vaunted, is often wanting, and, besides, there is little or no haemorrhage if, as many authors advise, the swelling is first constricted by the cold wire for a few moments and then cut through slowly and with pauses.

2. RHINITIS ATROPHICA (OZAENA).

Etiology and Pathology.—The origin of atrophic rhinitis, especially that form which is accompanied by foetor (ozaena), is still a question waiting to be solved. Many authors consider this form to be the second stage of hypertrophic rhinitis; the previously hypertrophic mucous membrane undergoing atrophy, at first at isolated spots, and then, later on, in its entirety, involving also the osseous and cartilaginous skeleton of the nose in the retrogressive metamorphosis.

Others again see in ozaena, a special infectious disease caused by a specific organism, the "ozaenococcus," and if this be so, the foetor must then be caused by a mixed infection. *Zarniko* believes that ozaena is due, not to an extraneous but to an intrinsic cause, namely, to a trophoneurosis. *Grünwald* denies the genuine character of rhinitis atrophica foetida, and holds that it is a symptom of suppuration of the neighbouring cavities or pharyngeal tonsil (focal theory of *Grünwald*).

Another series of investigators seek to solve the problem by biological methods, and trace the atrophic rhinitis to failure in development of the primitive organ; or to congenital abnormalities, *e. g.*, congenital smallness of the conchae, diminished resistance of the mucous membrane, metaplasia of the epithelium, etc. *Siebenmann* and *Meisser* direct attention to the formation of the face, and, according to them, it is due to the broad flat nose (platyrrhinia) usually met with in persons with broad faces (chamaeprosopia) in connection with the congenital metaplasia of epithelium. Besides these theories referred to, there are others, which aim at explaining pathogenetically some one or other of the prominent symptoms.

Some of them, however, do not at all explain the fact that ozaena sometimes occurs unilaterally. The great number of theories, which are often sharply opposed to one another, even though each may contain a grain of truth, show that none of them is free from objection. For this reason, and because of my long experience, I have come to the conclusion that ozaena is not a primary single disease, but manifests itself as a complexity of symptoms which is produced by a *variety of pathological processes on the substratum of predisposition; e. g.*, suppuration of the neighbouring cavities, primary hypertrophy, syphilis, etc., or congenital anomalies.

In this connection belongs also a kind of artificial atrophy, which is not so uncommon and is seen only after extensive resections of parts of the conchae or after repeated cauterizations; but the offensive smell, however, sometimes observed in these cases, is probably due to micro-organisms. *Frese* says that the micro-örganisms concerned are the same as those producing decomposition of albuminoids. The same author found substances in the secretions arising from atrophic rhinitis, which occur as the result of decomposition of albuminous matter, namely, indol, skatol, phenol, sulphuretted hydrogen, and a great quantity of volatile fatty acids. Of the same result was an examination of "ozaenous" scabs in a case of tertiary syphilis. We can therefore perceive that the "ozaenous" smell is not a unique or specific one, but a "bouquet" of various perfumes. This view is also held by *M. Schmidt.*

Whether we are justified in assuming a "*morbus sui generis*" in those infrequent cases in which we totally fail to recognize a cause, I do not venture to answer, and as the matter now stands, the assumption of a trophoneurosis would, in my opinion, certainly meet it best, though by this hypothesis not much is gained.

The confusion which still exists concerning the origin and nature of atrophic rhinitis might perhaps have its reason in that we do not know much concerning its actual commencement and course. It is a slow and insidious process, which comes under observation usually for the first time at puberty, when already all the symptoms are fully manifest. There are numerous observations on record that the disease may have already begun in early childhood. I myself was able to ascertain the existence of a pronounced ozaena in a child of two and one-half years, and an older sister, and the mother as well, suffered from the same disease. This circumstance, as also the observations of other authors, tends to show that heredity participates among the causes. Ozaena is more often met with in the lower classes, who live under more unfavourable and unhygienic conditions, than amongst those classes of better worldly circumstances. Females also suffer more commonly than do males.

Anatomy.—Both sides are usually affected, rarely one side only. It is often the lower conchae which is alone atrophied, while the middle may even show hypertrophy. In the advanced cases only, fragments of the conchae may be left behind. The mucous membrane is pale and yellowish, or gray and more or less thinned, as if it had been shrunk by adrenalin.

All parts of the mucous membrane become atrophied; the normal epithelium is often changed into that known as stratified and corneous in the superficial layers; the glands undergo fatty degeneration; the cavernous tissue shrinks, and the osseous structures also share in the general atrophy. At first there is round-celled infiltration, which is later on followed by the formation of fibrous connective tissue. *Ulceration never takes place.* It is essentially a chronic inflammation, leading to sclerosis of the mucous membrane. The atrophy of the periosteum and bone can be explained by the accompanying endarteritis and endophlebitis obliterans.

The secretion is very scanty. In the fresh state it is creamy and yellowish-green. It soon dries and then forms dull green, yellowish-green, blackish, or, through admixture of blood, brown or reddish-brown, hard scabs, which stick fast to the mucous membrane, like wall-paper, or are moulded and firmly wedged in the lumen, forming real casts of the cavity; on the mural surface they often show, however, a purulent film.

The secretion may be—this is certain—without any smell, but mostly, and, as a rule, all the cases with the marked formation of scabs, possess a smell difficult to be described, but which, once experienced, will never again be forgotten. From this smell alone the diagnosis is often made. It is this kind of atrophic rhinitis which commonly goes under the name of ozaena ("stink nose"). A better and more refined term is *rhinitis atrophica foetida,* in contradistinction to that kind of atrophic rhinitis which shows very little or no malodorous secretion. Simple atrophic rhinitis often affects the anterior part of the cartilaginous septum (locus Kiesselbachii), and produces there a site of predilection for "nose-bleeding" (rhinitis anteriora sicca). After repeated haemorrhages, the mucous membrane is often imbued with blood there, resulting in a sort of pigmentation of a dirty yellow or rusty color, described by *Zuckerkandl* as xanthosis of the nasal mucous membrane. A small amount of secretion always remains and dries, and forms small crusts there, giving rise to itching or burning, and, if these crusts be picked off, bleeding sets in.

Xanthosis may be found, though less often, on other parts of the mucous membrane besides the locus Kiesselbachii.

Symptoms.—The patients, though their noses may appear abnormally wide, with little or no secretion, complain of chronic obstruction It is often an easy matter to ascertain that the nose is perfectly free and pervious to the air-current, and that the sensation of blocking arises from the dryness in the nose simulating obstruction. This dryness is distinctly felt in the throat (pharyngitis sicca). Others again complain of fullness in the forehead, of headache, deficiency of or loss of the sense of smell. The sometimes obtrusively offensive smell, if suppuration of the neighbouring cavities is not present, is scarcely perceptible to the patient himself; but is more so to persons associating with him, who think that it comes from the patient's mouth. Only a professional examination shows that the smell comes from the nose, and not from bad teeth or the stomach. Many patients, on account of this horrible foetor, avoid society and become depressed. Sometimes the patient himself reports that smaller or larger crusts or scabs are removed from the nose, while endeavouring, not without difficulty or bleeding, to clear his nose. On external examination the broad, yet relatively small and low, nose, which looks as if it had prematurely ceased growing, with its point directed upwards ("saddle-nose"), is very noticeable. If we approach the patient the smell becomes more obtrusive and always indicates the formation of crusts. Indeed, if we introduce the speculum, we find one or both nostrils filled with crusts and scabs, which can only be removed with difficulty. This done, the nasal passages appear wide; and the upper margin of the choanae, the posterior wall of the pharynx,—sometimes covered with pus or scabs,—the mouth of the Eustachian tubes, and the prominence of the levator veli palati, ascending during speech or swallowing (prominence of *Passavant*), and in rare cases, the anterior surface of the sphenoid bone, can be clearly seen. Less obvious is this spaciousness in simple or partial atrophic rhinitis. The middle concha presents a peculiar appearance, which, with its bulbous anterior end (operculum), looks not unlike the edge of a blunt-pointed knife. Small scabs are often seen covering the anterior end of the concha like a cap. The evil smell is much lessened or reduced after removal of the scabs or crusts.

Diagnosis.—Whenever we find a more or less marked shrinking of the mucous membrane and bone, and, in connection with it, a more increased wideness of the nasal cavity, we are justified in diagnosing simple atrophic rhinitis, and if we encounter foetor ex naso, rhinitis atrophica foetida. To make the diagnosis solely on the foetor alone seems to me risky, though it is often characteristic, for the same odour may arise from suppuration in a neighbouring cavity from foreign bodies or ulceration within the nose. But in these cases it does not disappear after removal of the discharges and secretions, as it does in atrophic rhinitis; and the atrophy, which is so characteristic of rhinitis, is wanting. If, on the other hand, these processes are of long-standing duration or they have been cured, the picture that remains is not any different from that which we see in uncomplicated atrophic rhinitis; and it is then quite justifiable to speak clinically of a simple or foetid atrophic rhinitis. But if one conceives rhinitis atrophica foetida (ozaena) as a disease *sui generis*, then this term must then only be applied to those cases where any other cause cannot be found.

Ulceration of the nose in connection with atrophy is nearly always due to syphilis.

Prognosis.—Inveterate cases of simple (or genuine) atrophic rhinitis are incurable, because a restitution of the destroyed regions is impossible. The loss of smell is often permanent. Slighter cases are, though frequently only transitorily, amenable to cure. With advancing age the foetor and the scabs frequently disappear of their own accord.

Treatment.—Here, our first task is to remove the evil-smelling scabs and to prevent the re-occurrence of fresh ones. On first examination we remove all scabs as much as we are able to with forceps, even if it be only for the purpose of ensuring a diagnosis. Later on the removal of the scabs must be done by irrigation of the nose with a nasal douche. We must irrigate with a lukewarm solution of common salt (sodium chloride) or bicarbonate of soda, borax, or boric acid. For the technique of the proceeding see p. 38; and the douching must be done twice daily, and oftener in more severe cases. These irrigations soon result in the relatively quick disappearance of the terrible foetor. We may increase the effects of the irrigations by putting into the diseased nostril small pledgets of cotton-wool, and if both sides are diseased, into both nostrils. With a little practice the patient himself learns to push into the nose pads of cotton-wool 4 to 5 cm. long, which are rolled in the form of a cigarette, so far backwards and upwards that the lower part of the nasal cavity remains free for respiration. This is the so-called *Gottstein's* method of plugging. (See p. 37.) The size and thickness of the rolls depend on the width of the nose, and care must be taken that the pad does not press too much on the mucous membrane. To introduce the rolls we may use the forceps, or the wool is wound on a *Gottstein's* tampon-holder. Having pushed the plug far enough into the nose, the instrument can be withdrawn by twisting it in the reverse way. The roll may remain for several hours—sometimes a quarter of an hour is quite sufficient to loosen the scabs, so that, by the act of sneezing, they are discharged. Instead of plugs of cotton-wool *Franz Brück* recommends small strips of folded gauze, which should remain so long that they are thoroughly moistened by the secretions. *Gottstein's* method of plugging, consistently performed, alternating on the one side and then the other, or on both sides, is often sufficient to prevent the re-formation of fresh scabs; and through the narrowing of the lumen by the plugs, the drying and exsiccation of the secretion is also impeded. More recently submucous injections of paraffin have been tried in order to produce narrowing; and in certain cases improvement and even cure are reported. The cupping (congestive) method, after *Bier* (pp. 23, 24), energetically applied, is reported to have had good results.

In any case, the application of the tamponade, in combination with douching, has given valuable results, and in a short time the scabs and foetor will disappear. Massage of the mucous membrane is also beneficial; and this is done by the method referred to on p. 40. For the lubrication of the dressed tampon-holder, liquid paraffin or glycerin may be used (p. 37), or any indifferent ointment might serve the same purpose, as also in *Gottstein's* method. We can massage daily, or every alternate day, for two minutes at a time.

Besides local treatment, we must also treat the entire constitution; as, for instance, by ordering iron, arsenic, or iodide of iron, suitable diet, etc. If the disease of the nasal cavity is only the sequel or complication of a disease in a neighbouring part, this latter must be treated beforehand.

VI. SPECIAL FORMS OF RHINITIS.

1. ULCUS SEPTI PERFORANS (PERFORATING ULCER).

Ulceration may occur in the septum as a symptom of a general disease, and be due to tuberculosis, syphilis, glanders (malleus), etc. They often lead to perforation of the cartilaginous septum. Inflammation of the mucous membrane may, however, in due course extend in depth and into the cartilaginous septum, so causing ulceration and perforation (idiopathic ulcer; ulcus septi perforans). The rhinitis sicca anterior before described not infrequently leads—if the patient, from constant irritation, picks the nose and thereby injures the mucous membrane—to an infective inflammation, which slowly and gradually spreading, in time causes necrosis and finally perforation of the septum. The perforation shows sharp, and, as it were, pared away and thinned edges. In certain trades perforation is conduced to by local nasal irritation (workers in dye, in cement, chemical works, etc.). Perforation does not cause much inconvenience, and is often only incidentally discovered.

Diagnosis.—Diagnosis depends on the typical site of the ulcer on the cartilaginous septum, on its round or oval shape; further, the signs of inflammation are usually absent.

Syphilitic ulcer is not, or very usually not, limited to the cartilaginous septum, and there are mostly other signs of syphilis. The ulcer itself is covered with a grayish film, and is easily influenced by mercury or iodides, whereas the idiopathic ulcer reacts very little to the application of these special drugs. Tuberculous ulcers show granulations and undermined edges.

Ulceration or ulcers having been cured, distinction between the various kinds of ulcers often becomes impossible. We may also mention that perforation sometimes follows an operation for deviations of the septum.

Treatment.—As long as perforation has not actually taken place, treatment consists in cauterizing the ulcer with lapis infernalis, and applying a neutral ointment. After perforation has really occurred we must limit our action to douching and anointing. Prophylactic measures consist in removing, as much as possible, any harmful irritation. Picking the nose must be strenuously forbidden, and itching can be relieved by anointing the parts with the following ointment: Ung. diachyl.; hydrarg. oxyd. (hydrarg. oxyd. flav., 0.2; vaselini, 10.0; ft. ung.).

2. HAY-FEVER (BOSTOCK'S DISEASE).

Also called *Bostock's* catarrh, after *Bostock*, who first described it. Is still sub judice in regard to its pathology.

Symptoms.—It makes its appearance under the clinical picture of a violent cold, though not necessarily with fever. Its most prominent symptoms are severe conjunctivitis, obstruction of the nose, copious watery discharge, and paroxysms of sneezing. In more severe cases, which may develop from slight ones after repeated attacks, the catarrh spreads to the other air-passages, the larynx, trachea, and bronchi, causing difficulties in breathing; and the so-called "hay asthma." Hay-fever occurs periodically, when the grass, flowers, and the corn bloom (say, from May to end of June), and it only attacks persons who are specially predisposed to it or who are overworked or run down in their general health. The attacks last from a few hours (which is seldom) to five or six weeks.

On rhinoscopic examination we find nothing characteristic of the disease. During an acute attack the mucous membrane is swollen and much injected.

Etiology and Pathology.—*Helmholtz* maintained that it was an acute specific disease, due to specific germs. His opinion, however, does not find many adherents. The general trend of opinion of present observers is that hay-fever is some kind of rhinitis on a nervous basis, and an attack is provoked by the irritation caused by aspiration of graminaceous pollen.

Dunbar and his pupils, in a long series of investigations, have shown that we here have a specific disease, viz., that it is a poisonous proteid matter which is contained in the starchy rods of the pollen of certain gramineae,—the so-called pollen toxin,—which produces the characteristic symptoms of hay-fever. According to *Dunbar's* theory, hay-fever would thus represent an idiosyncrasy of certain persons towards the pollen toxin of various plants; indeed, a specific poisoning. In America, where the disease is much more common than in our country, there are some plants which blossom in the autumn, and which cause the disease known as "autumn fever" (catarrhus autumnalis).

Diagnosis.—The diagnosis depends on the periodicity of the symptoms. Difficulties may arise if we have to discriminate between "hay asthma" and ordinary bronchial asthma. Nocturnal attacks of asthma, however, are generally bronchial; attacks during the day, on the other hand, may be "hay asthma." In doubtful cases we may try to provoke an attack by exhibiting pollen toxin.

Prognosis.—Prognosis is, *quoad vitam,* always favourable; less so as regards ultimate complete cure.

Treatment.—*Dunbar,* on the ground of his pollen theory, has indicated a so-called specific treatment. From the lymph of animals, who have been treated with pollen toxin, he has made a hay-fever antitoxin, which is used under the name of "pollantin," either liquid or powdered. *Dunbar* advises that the powder as a preventive should be brought with a brush in contact with the conjunctiva of the lower eyelid in the early morning, in very small doses. Also that a small quantity of the powder, about the size of a lentil, should be sniffed up or put into the nose with a small scoop, or, instead of the powder, the fluid preparation may be used. "Pollantin" has encountered a varied reception, and many favourable reports are at hand; but at present a final judgment is impossible. *Weichardt* has brought another serum on the market, called "Graminol," which he makes from the normal lymph of graminivorous animals at the time of the blossoming of the gramineae. According to *Wolff-Eisner,* the pollen toxin is not a real toxin, but merely a proteid, which, like all proteid toxins, has the quality of producing an increased "receptibility," but no immunity and no formation of antitoxin. The effect of the hay-fever serum, respectively, of pollantin and graminol, is, therefore, neither specific nor antitoxic, "*but is that of a colloid body, like the antiprecipitins.*"

If the resources of the patient allow it, we send him, during the critical season (May–June), to a place free from pollen dust (Heligoland, St. Blasius in the Black Forest, Oberhof in Thuringia, Gastein in Salzburg, Cortina in the south of Tyrol) or on a sea-voyage. Or, on the other hand, he must stay in his rooms, with the windows shut, as long as it is not unpleasant, and avoid flowering meadows, and should, if he walks in the open air in the neighbourhood of meadows or corn-fields, wear a protective apparatus designed by *Mohr* (see Fig. 42), which is really only *Schmidthuisen's* nasal wing elevator screened with cotton-wool to filter off the impurities of the air.

Denker, Urbantschitsch, and others, having the idea that a local disposition is necessary in order to acquire hay-fever, desire to diminish this presupposed irritability of the mucous membrane by massage à la *Gottstein* or by an electric vibrator. The same object, however, can be carried out by painting the mucous membrane with cocaine or adrenalin.

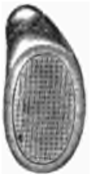


Fig. 42.—Nasal air filter (*after Mohr*).

We cannot recommend operative manipulations, such as cauterization or galvano-cauterization, for they are not only useless, but positively injurious, for the reason that they render the patient more nervous than he was before.

It is of great importance to strengthen the patient's general health by general treatment (hardening, and forbidding alcohol and the excessive use of tobacco).

VII. ACUTE AND CHRONIC INFECTIOUS DISEASES.

1. ACUTE EXANTHEMATA.

Of the acute exanthematous diseases, scarlet fever and small-pox mostly, and measles always, are accompanied by an acute catarrh of the nasal mucous membrane. The catarrh often, especially in scarlatina, spreads to the annexed cavities. *Koplik's* spots, so characteristic in measles, make their first appearance, according to *Catti,* in the nose, and not in the mouth; and often in conjunction with bleeding. In enteric fever in young patients epistaxis is a frequent symptom. Generally speaking, the nasal catarrh is only part of the general infection, and, if the accessory cavities are not seriously implicated, there is no need of special treatment.

2. INFLUENZA.

In "la grippe" or influenza we have to deal clinically with an acute nasal catarrh. This catarrh of influenza, however, shows such marked symptoms of its own that we have to consider it separately. Here complications of the accessory cavities, mostly of the frontal sinus, and violent neuralgias (headache and frontal neuralgia) are very common. These latter are often the accompaniments of the implication of the accessory cavities, or may be due to inflammation of the nerves (peripheral neuritis). Partial or complete loss of smell and epistaxis complete the picture. After the catarrh has subsided, the neuralgia may still continue to exist for a long time.

Treatment can be only symptomatic, as in other cases of coryza.

3. GONORRHOEA.

Rhinitis gonorrhoica (blennorrhoica) is rarely met with in adults, not so seldom in infants, who have been infected during their entrance into the world by the vaginal secretions. It occurs always in conjunction with gonorrhoeal conjunctivitis (blennorrhoea neonatorum). In such cases the muco-purulent secretion is very copious and will mostly be found to contain gonococci.

Treatment.—Treatment must follow the same lines as for other acute catarrhs.

4. RHINITIS DIPHTHERICA (FIBRINOSA).

Etiology and Pathology.—Diphtheria of the nasal mucous membrane is in nowise different from pharyngeal diphtheritic inflammation, for it is, in the great majority of cases, only a part of the whole disease. Diphtheria of the pharynx may be primary, and then the disease spreads into the nasal cavity, secondarily, or, what is probably less common, the disease begins in the nose primarily and from here invades the pharynx.

Scheller and *Stenger* think that the beginning of the disease in the nose is, indeed, common enough. In some cases, infection of the entire body takes place through the nose without affecting the latter. There the bacilli may remain quiescent or dormant for a long time, and only begin their dreaded work, if there has been formed a locus minoris resistentiae—in a case reported by *Scheller* and *Stenger* an intranasal operation was followed by it.

It is different with the fibrinous (croupous) form of rhinitis, which is regarded by many authors as a mitigated form of diphtheria caused by a primary infection of the nasal mucous membrane by the *Klebs-Loeffler* bacilli. It is of a local and benign character, and reminds us of those cases of mild pharyngeal diphtheria where the disease does not cause general symptoms and is limited to the tonsils alone. We should like, however, to point out that the diphtheria bacilli have not been found in all cases of fibrinous nasal inflammation—according to *Gerber*, in 75 per cent. of all the cases, the bacilli found have shown themselves to be of low virulence; and also that other organisms, such as staphylococci, were present.

On the weight of this evidence we may reasonably conclude that infection by diphtheria bacilli may be secondary to that caused by others. This circumstance finds its analogy in that we often find other pathogenic micro-organisms, *e. g.*, pneumococci or meningococci, in the company of *Klebs-Loeffler's* bacilli, which latter seemingly assume only the rôle of saprophytes in relation to their human host. Those cases of fibrinous rhinitis take a separate rank where the formation of fibrinous membranes is produced by chemical or galvanic cauterization.

I think, after all that has been said, we are on the safe side if we do not classify, for the time being, fibrinous rhinitis as a distinct disease; at least we must recognize a mild and a virulent form; and in practice it is safe to make no distinction at all, and to isolate each case of fibrinous rhinitis as long as we find, on microscopic examination, that *Klebs-Loeffler's* bacilli are present.

Both variations are met with mostly in children.

In infants we sometimes observe a kind of diphtheritic inflammation which is not caused by *Klebs-Loeffler's* bacilli, but by streptococci, *i. e.*, through septic infection from the maternal genitals during the puerperium or from dirty and infectious linen.

Symptoms and Course.—Fibrinous rhinitis, begins like an ordinary acute coryza, fever not always being present. The discharge, at first mucous, becomes, later on, muco-purulent and blood-stained, and leads quickly to obstruction of both sides—seldom of one side only—of the nose. On examination we find in the nose a grayish, or yellowish, gelatinous, more or less thick, false membrane, which adheres firmly to the mucous membrane, and cannot be separated from it without great difficulty. Any false membranes removed are soon reproduced. The process of recovery usually takes several weeks; and the false membrane ceases to form the sooner the less we disturb it.

True *diphtheritic rhinitis* begins also with the symptoms of an acute catarrh, and in certain mild cases may stop short at this. The true character of the disease is only discovered by the occasional discharge of false membranes or through microscopic examination (*Klebs-Loeffler's* bacilli). But mostly, from the very first onset, the general health is greatly disturbed: the nose is blocked; the copious secretions, at first watery, later on muco-purulent, cause erosions at the nares and on the upper lip. The nose bleeds freely, and grayish false membranes are discharged with the act of sneezing or blowing. In cases where a mixed infection has taken place (staphylococci and streptococci, besides diphtheria bacilli), the mucous membrane may become gangrenous. The broken-down and necrosed shreds or false membranes have a horribly foetid odour.

In secondary nasal diphtheria the symptoms are, from the onset, masked by the pharyngeal disease.

Diagnosis.—In all doubtful cases a microscopic examination should be made and the character of the disease proved by the presence or absence of *Klebs-Loeffler's* bacillus, though, as we have seen, it is not an absolute test. The clinical features of the disease are also deceptive, save in cases where the symptoms have reached their acme or the rhinitis is secondary to an original faucial diphtheria or a primary rhinitis spreads and invades the pharynx.

Prognosis.—In the simple fibrinous (croupous) form the prognosis is mostly good, but it is serious in diphtheritic inflammation.

Treatment.—In *fibrinous rhinitis* following chemical or galvanic cauterization all local treatment should be stopped, and, in other forms of the disease also, the mechanical removal of the fibrinous membranes should altogether be avoided. In diphtheritic rhinitis all that it is necessary to do is to apply locally, every two or three hours, small tampons of cotton-wool soaked in salicylic oil, 1 per cent., or menthol oil, 10 per cent. That sometimes relieves the nose, and painting also with adrenalin will sometimes free the nasal passage; then afterwards a 4 per cent. solution of boric acid or 0.1 per cent. of permanganate of potash, in order to deodorize, may be cautiously injected. Unfortunately, the local treatment does not often have the desired result. In the non-septic cases, we have it in our hands to avert dangerous consequences by the early injection of antidiphtheritic serum. There exist two sera, a simple one and one of high value, the application of which is guided by the degree and period of the disease and by the age of the patient. Generally, it is best to inject at once the larger dose, and to repeat the injections in order to saturate the toxin circulating in the blood and to neutralize that which has already become absorbed into the various organs.

The best place for an injection, which is sometimes followed by a rash, is the chest or thigh.

Local treatment is not rendered superfluous by the injections of serum. Great attention must be given to the general treatment, especially to the condition of the patient's heart, which often requires stimulants.

It goes without saying that the patient should be isolated, and the discharge and secretions of the nose and mouth be made innocuous as quickly as possible.

5. TUBERCULOSIS AND LUPUS.

Tuberculosis and lupus of the nasal mucous membrane are relatively rare. Though both are caused by *Koch's* bacillus, clinically they show a different course. How this difference is caused, and whether the tubercle bacillus is modified by the secretions of the mucous membrane, is not yet decided. Tuberculosis of the nasal mucous membrane is usually secondary to that of the lungs or larynx, but it may also occur primarily in the nose, as when the mucous membrane has been infected by dirty linen or by the finger.

Lupus of the mucous membrane is always associated with lupus of the skin. It is then difficult to say which is the primary seat of disease. It is a curious fact that women more often suffer from lupus of the nose than men.

Etiology and Pathology.—The essential feature of tuberculous disease or lupus is the tubercle, a little gray or yellow-gray nodule. The nodules soon break down, forming ulcers, which may coalesce to form larger ulcers. Or they give rise to infiltrations; rounded or oval, reddish infiltrates, which appear as circumscribed, elevated, easily bleeding, friable papules (tuberculoma). The infiltrations and tuberculomata soon break down, however, and form large ulcerated areas.

The nodule of lupus is the same thing as the tubercle, but it has less tendency to break down, and not infrequently atrophies.

On microscopic examinations one finds round-celled infiltration and giant-cells, especially around the blood-vessels. Tubercle bacilli, however, are generally scanty.

Symptoms.—At first there is little to be seen; later, if infiltration and tuberculomata have grown up or ulceration supervenes, the nose is obstructed, and there is a copious purulent, blood-stained discharge, with ample formation of crusts and scabs. The disease causes little pain, and the general comfort is little disturbed, except other important organs, such as the lungs or larynx, become affected. The disease is so insidious and unobtrusive that patients do not usually consult a physician unless ulceration has occurred. Then one finds ulcers covered with crusts or scabs at the nares, on the septum, or perforation of the septum. On removal of the crusts, irregular notched ulcers can be seen, with their edges thickened, corrugated, undermined, and covered with granulation tissue. In the adjacent mucous membrane miliary tubercles are visible.

The skin of the nares and upper lip sometimes shows inflammatory infiltration and is covered with crusts; conjunctivitis, from obstruction of the naso-lachrymal duct, makes its appearance; and the cervical and submaxillary glands become enlarged—signs which are often summarized under the term "scrofula."

But in truth, they are really a distinct form of infantile tuberculosis, and the term is often erroneously applied to those cases where a chronic nasal catarrh with copious discharge, eczema, and infiltration of upper lip and nares, and swelling of the glands, is associated with adenoid vegetations.

In lupus of the mucous membrane we see nodules of the size of a millet seed, often covered with crusts and arranged singly or in clusters; at a later period ulceration has occurred.

In many cases there is little or no difference between tuberculosis and lupus; the latter shows more tendency to shrink and atrophy, and the former tends more to break down. If we desire to be safe, we will confine the diagnosis of lupus to those cases where the affection of the mucous membrane is associated with lupus of the skin of the nose or face, or where we see the nodules so characteristic of lupus. As regards our procedure, there is no difference if it be either lupus or tubercle.

Prognosis.—Prognosis seems to be somewhat better in lupus. Relapses are frequent in both diseases.

Diagnosis.—Diagnosis will be easy if the skin of the nose or face is also diseased. Tubercle at its commencement might occasionally be mistaken for lymph follicles. These, however, are nothing but an accumulation of lymph cells, which have no inclination to break down. Tuberculomata, if they are very large, may simulate malignant tumors (sarcoma), and in such a case the microscope will soon settle the question. Greater difficulties arise in the diagnosis between tuberculous and syphilitic ulcer. *Tuberculous ulcers are in general much more indolent than syphilitic ulcers.* In the former the adjacent mucous membrane is little inflamed or affected, the discharge smells little or scarcely at all, and pain is mostly, if not always, absent. *Syphilitic ulcers* are always surrounded by a wall of inflammation; the discharge is a beastly smelling secretion, and there is often acute neuralgia in the area supplied by the trigeminal nerve. Tuberculosis is mostly localized and limited to the cartilaginous septum; syphilis frequently affects the osseous septum. Diagnosis is aided by other signs of the respective disease in other organs, by the microscopic examinations, or *ex juvantibus*, by the effect of iodine. But it should not be forgotten that both diseases may occur jointly in the same place.

The idiopathic ulcer of the septum shows smooth, thinned edges.

Treatment.—Isolated granulomata (tuberculomas) can be removed, after previous local anesthesia, by the snare; broadly seated ones, as well as ulcers, must be energetically scraped out, down to the healthy tissue, with a sharp scoop, and then cauterized with lactic acid (50 to 80 per cent.) or the galvano-cautery. *In lupus the hot-air treatment* may be highly recommended. In advanced cases, where the general health is also affected, treatment should aim at cleaning the ulcerations. In regard to the treatment of lupus of the skin, we refer the reader to the text-books on skin diseases.

Local and general treatment must always go hand in hand.

6. SYPHILIS.

Following the usual grouping, we also find in the nose primary, secondary, and tertiary affections; the various forms, however, cannot always be sharply separated from each other, and often pass insensibly from the one stage into the other, or are concomitant.

(*a*) The **primary affection** (initial sclerosis, hard chancre) is not often found on the nose, more often at the entrance or on the septum of the nose; that is, on those places where a transmission of the syphilitic virus, through the finger, kiss, or handkerchief, etc., is made easy; or it may be found in the naso-pharyngeal space, near the ostium of the Eustachian tube, through infection from unclean catheters.

Hard chancre of the introitus is characterized by a hard, indurated, flat infiltration showing a shiny, red, smooth surface under a thin crust. It soon breaks down into an ulcer with hard infiltrated edges and scanty discharge, and is covered with a shiny grayish or yellowish débris. On the septum or naso-pharyngeal space chancres appear as red, hard, raised infiltrations covered with a shiny coat. Corresponding to the seat of the primary affection, the nose may be swollen and obstructed; headache and fever may be present, or, in retro-nasal chancre, diminution of hearing. The regional lymphatic glands (cervical and submaxillary) are always swollen.

(*b*) The **secondary stage,** as elsewhere, is characterized by the two types of secondary syphilis, viz., erythematous and papular eruption. This shows itself in the nose as syphilitic catarrh and condyloma; the latter, of very rare occurrence.

Syphilitic coryza, clinically, is very little different from the simple, non-specific variety, and is, for that reason, often not recognized, but lasts much longer. This obstinacy is also very marked in specific catarrh of infants—so-called snuffles of the new-born (coryza syphilitica neonatorum) which, in *Lesser's* opinion, no child affected by hereditary syphilis escapes. Every long-lasting cold in a physically badly developed infant, especially if accompanied by blood-stained secretion and discharges of crusts and scabs from the nose, is indicative of syphilis. Later ulceration occurs, followed by necrosis and defects of the osseous structure of the nose (saddle-nose). It is of no consequence, if one attributes these manifestations of the secondary or tertiary stages to the result of gummatous ulceration, for the treatment is very much the same. These advanced lesions are sometimes observed in much older persons, the eruptions and manifestations of earlier youth having passed over without leaving a trace.

The papular syphilide and broad condylomata at the entrance of the nose appear as small brown or brownish-red patches or papules. They tend to break down and lead to painful cracking of the tissues (fissures and rhagades), mostly in the posterior angle. On the mucous membrane they show themselves as gray or yellow opacities in the epithelium, chiefly on the mucous membrane of the septum and floor of the nose.

(*c*) The most common manifestations of syphilis in the nose belong to the **tertiary stage.** They are represented by the gumma or gummatous infiltrations, and are of a more diffuse character, or are more or less circumscribed. Microscopically, one finds a small-celled infiltration. The gumma originates either in the mucous membrane or grows from the periosteum or perichondrium of the nasal skeleton. It leads, by necrosis, to extensive destruction if it be not interfered with. The gumma of the mucous membrane soon breaks down and forms an ulcer, the floor of which is gray and slimy; its edge is punched out, indurated, and sharply cut. It invades the underlying structure, cartilage or bone, or both, according to its position. If it is primarily seated in the bone or cartilage, this will be destroyed by necrosis and atrophy, and is discharged as sequestra. In this case, the mucous membrane will be destroyed from within outwards.

On the other hand, syphilis may lead to sclerosis and hyperplastic processes of the bone, in particular of the ethmoidal bone.

Symptoms and Course.—The beginning of the tertiary stage frequently escapes observation. The patients are seen when ulceration and sequestration have taken place; then the nose is obstructed, and especially if the bones are affected; discharges, foul-smelling pus, and blood-stained secretion, headache and neuralgia are common and unpleasant additions. The septum is the most common seat of tertiary syphilis, and ulceration of the osseous septum can be considered as pathognomonic. It is likely to spread in depth and to perforate the bone, which is destroyed and ultimately discharged in the form of a sequestrum. Cases are recorded where the whole septum had been destroyed, yet, nevertheless, the nose preserved its external shape for a long period. Sinking-in of the bridge of the nose is not caused through the loss of the septum, which really does not support it, but through the cicatricial retraction and shrinking in at a later stage of the fibrous tissue, which pulls and retracts the cartilages and mucous membrane connected with the nasal bones. In this way the disfigurement so often seen in syphilitic persons, called "*saddle*" and "*lorgnette*" *nose,* must be explained. A combination of both deformities is the so-called "bull-dog" nose, where the nose is, so to say, withdrawn into the pyriform nasal aperture. (See Fig. 43.)

Besides being on the septum, we also find gummata on the floor of the nose, where they form spherical swellings tending to ulceration, and finally to perforation of the hard palate. In such a case speech becomes nasal in sound, and not infrequently food may be driven into the nose during the act of chewing. The conchae may suffer in the general destruction, which may become very serious if the neighbouring bones are implicated, and the disease spreads into the interior of the skull, causing meningitis.

The skin of the outer nose, particularly that of the alae nasi, may be affected and destroyed by gummatous infiltration. In this way adhesions (synechiae) and constrictions (stenoses) of the nasal cavities occur, and in some malignant cases, when

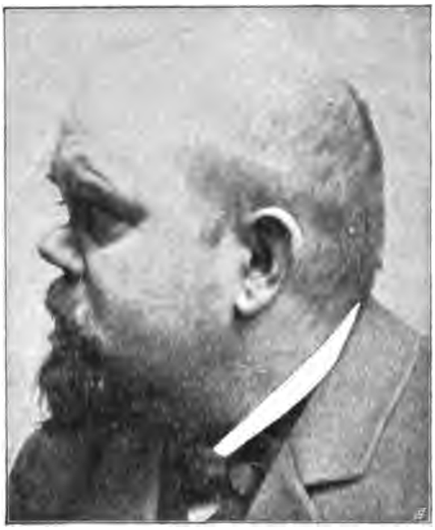


Fig. 43.—Syphilitic saddle-nose.

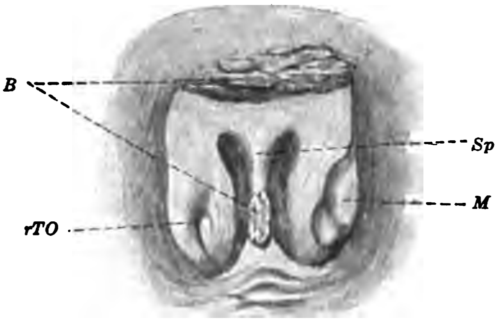


Fig. 44.—Syphilitic saddle-nose, seen by anterior rhinoscopy through the left nostril. The septum is entirely destroyed, and only a small splinter of bone is left (*Sp*) between the choanae. The whole nasal cavity can be seen from each nostril. A fragment of the left inferior turbinate remains (*M*), which covers the left ostium of the Eustachian tube, seen from the left nostril; the right tubar ostium (*rTO*), with the tubar wall, is very distinctly visible. Splinter of the septum and roof of the nose covered with scabs (*B*) showing atrophic post-syphilitic rhinitis.

both external and internal parts of the nose are diseased, the ensuing destruction may be of so great an extent that, of the whole nose, nothing but the cavity remains.

It sometimes happens that the process comes to an end by itself, after the discharge of all the necrosed parts. Usually the result is atrophic rhinitis. The fibrous elements of the mucous membrane undergo hypertrophy and hyperplasia, which, in their turn, give place to retrogressive metamorphoses, shrink, and atrophy. The secretions dry up and form scabs and crusts, and assume the same odour as in "genuine ozaena" (rhinitis atrophica syphilitica foetida) (syphilitic ozaena).

In a third class of cases no scabs are formed, and the very spacious nasal cavity shows only the signs of simple atrophic rhinitis.

On rhinoscopic examination we find a most varied picture. Rarely do we see the infiltrations, mostly ulcers, covered with crusts and scabs on the parts aforementioned, diffusing an infamous odour. On applying a probe we encounter bone, denuded of its periosteum, rough, and ultimately movable. The mucous membrane itself is red and swollen, and in the later stages we find exostoses or adhesions (osseous or fibrous), or the nasal cavity forms a wide hollow space, which shows only traces of "decayed magnificence."

Diagnosis.—Primary chancre often passes unrecognized until the regional lymphatic glands are swollen and eruptions of the skin have made their appearance. In the secondary stage, the signs and symptoms in the nose are the same, and are always associated with those on other parts of the body. Tertiary syphilis is, for the most part, easy to diagnose, and more so the further advanced the process. In the first stage we may have difficulty in diagnosis, especially if anamnesis and objective examination are uncertain. Later, the destruction of the osseous nose and the odour will reveal the nature of the disease.

With regard to the differential diagnosis between tuberculous and syphilitic ulceration, we must refer to the previous chapter, and in doubtful cases a resort to iodide of potassium will sooner or later decide. Whether we are justified in relying on a bacteriological examination cannot yet be asserted with certainty.

The Spirochaeta pallida, discovered by *Schaudinn* and *Hoffmann*, is not yet generally accepted as the causa agens of syphilis.

Prognosis.—Prognosis is favourable if a specific treatment is undertaken. Unfortunately, there are cases which, in spite of the best treatment, assume malignant character; and others, again, which frequently relapse.

Treatment.—Treatment must be local and general. In the case of hard chancre it can only be local. If it is seated externally, it can be covered with a mercury plaster, or, if ulcerated, washed with a sodium chloride solution, and afterwards dusted with calomel. If it is found on the septum, or even farther in, the nasal cavity can be douched with a 0.1 per cent. solution of permanganate of potash or insufflated with calomel. The same treatment can be applied locally in the secondary stage. Fissures may be covered with white precipitate (mercury) or may be cauterized with chromic acid. The general treatment of secondary syphilis consists in the exhibition of mercury, either as inunction or intramuscular injection; or if either or both is inadmissible, then in the form of pills; as:

℞. Hydrarg. tannic. oxydul	2.0
Basic alum. silicate	6.0
Glycerin	q.s.

Ut f. pil. No. xxx.
SIG.—Two pills after meals three times daily.

In whatever form mercury is exhibited, the utmost care and attention should be exercised in keeping the mouth and teeth clean and in order, so as to avoid mercurial stomatitis, which is very apt to retard or complicate the treatment. The teeth should be put in order before the commencement of mercurial treatment. The whole oral cavity and the throat should be cleansed and washed as often as possible. As a mouth-wash or gargle the liquor alumini acetati diluted (*Burow's* solution), or perhydrol, 1:10, may be used. For the same purpose tincture of myrrh (20 drops in a tumbler of water) or a solution of permanganate of potash may be applied. The teeth must be cleansed after meals by the tooth-brush and powder. In the later stages, mercurial treatment is effectually aided by iodide of potassium. Infants must be bathed daily, for a quarter or half an hour, in a warm bath (a wooden bowl), to which 1.00 bichloride of mercury is added, while internally calomel is given. In tertiary syphilis iodide of potassium is the sovereign remedy. One gives it in solution of 8–10–15: 200, of which a tablespoonful in milk after meals is taken three times daily, and which may be suitably combined with a mercurial inunction. To persons who cannot stand the use of iodide of potassium, and who soon get coryza, or eruptions, the doses must be reduced, and an alkali added; still better in such cases is iodipin, an organic compound of iodine with sesame oil:

℞. Calomel	0.002–0.01
Sacch. lact.	0.30
Sig.—One powder three times daily.	
℞. Iodipin (10 per cent.)	100.00
Ol. menth. pip.	gtt. iij
M. Ft. Sig.—One tablespoonful three times daily.	

The general treatment, such as we have outlined, is in most cases sufficient; it may be supplemented with washing out or douching the nose, but local treatment is mostly unnecessary, if it be other than for the removing of necrosed bone. This, however, should be extracted only if it is completely movable; and also requires special skill, if it has to be removed from the roof of the nose.

The treatment of the various conditions, as synechiae, formation of scabs, and ozaena, is the same as in non-syphilitic cases, and we refer the reader to Chapter V (p. 82). Ensuing facial disfigurement (saddle-nose, bull-dog nose, etc.) can be rectified by the injection of liquid hard paraffin. This procedure is not always harmless, and its lasting effect doubtful, for the paraffin is slowly, but for the most part, absorbed.

7. RHINOSCLEROMA.

The disease, which is very chronic in its course, usually begins in the nose, but also occurs primarily in any part of the upper air-passages. It would, therefore, be better to speak generally only of scleroma, and to preserve the name rhinoscleroma for the affections of the nose solely.

The disease seems to occur mostly in eastern Europe, and in persons of middle age, living under imperfect hygienic conditions. It is probably caused by the "scleroma bacillus," discovered by

Frisch, which appears to be a short, thick bacillus, enveloped by a capsule, similar to *Friedländer's* bacillus.

Symptoms and Course.—The beginning is usually that of nasal catarrh, with a discharge of odourous secretions and formation of scabs. Rhinoscopically, the nasal cavity shows rhinitis atrophica. Gradually the nose becomes obstructed, and this is due to infiltration of the mucous membrane, at first circumscribed and nodular, later on becoming more diffuse, and situated at the commencement mostly in the anterior part of the floor of the nose, but oftener in the naso-pharynx. It then spreads on all sides to the external nose as well as the lip, face, and forehead. At first soft and red, the infiltration later becomes as hard as cartilage, pales, and undergoes atrophic processes, *e. g.*, shrinks and forms scars, producing adhesions, constrictions, and disfigurement. Finally, the nose appears thickened, like a bulb. There is never any suppuration.

In its further course the scleroma invades the naso-lachrymal duct, annexed cavities, throat, and other air-passages.

Microscopically, one finds round-celled infiltration interspersed with larger cells, which show colloid degeneration and contain the scleroma bacilli. These bear the name of *Mikulicz's* cells. In the later stages, new formation of connective tissue is conspicuous.

Diagnosis.—The diagnosis is based on the particular localization, on the extremely slow development, the conspicuous hardness, and on the absence of ulceration.

At the beginning it might be confused with sarcoma, carcinoma, or gumma, as long as the outer skin is intact. Sarcoma and carcinoma, however, are seldom bilateral, and gummatous infiltration is also mostly unilateral. In doubtful cases the trial of iodide of potassium or a probationary excision, with microscopical examination, would ensure the diagnosis. The distinction between rhinoscleroma and rhinophyma offers no difficulty. (See p. 54.)

Prognosis.—Prognosis is unfavourable, as the disease is incurable. If it spreads to the larynx, it is then directly dangerous.

Treatment.—This is hopeless. Transitory relief can be rendered by operative measures on the obstructing parts of the tumour, or by the introduction of bougies in order to prevent adhesions. If the latter have already occurred, they must be broken down by some method. More recently, *x*-rays and *Finsen*-light treatment have shown themselves very valuable, but it is doubtful whether this treatment has any lasting effect.

8. MALLEUS (GLANDERS; FARCY).

Glanders is an infectious disease, peculiar to horses, and is caused by the Bacillus mallei. It is sometimes transmitted to men who come into intimate contact with horses, as hostlers, grooms, stable helpers, etc. It is characterized by circumscribed and nodular, more seldom diffuse, infiltrations, which have a tendency to break down and form ulcers and abscesses. The bacilli are transmitted through fissures or erosions of the mucous membrane or skin, but the investigations of *Babes* show that infection can take place through the intact mucous membrane of the nose and mouth.

The disease is acute, chronic, or of great variety, as the case may be. The affection of the nose, albeit often rarely absent, is often insignificant. The nose is seen to be more or less swollen, as in erysipelas. There is a copious, purulent, evil-smelling discharge; and on rhinoscopical examination, one finds here and there little yellow papules or pustules which later on ulcerate. The ulcers show a hard, infiltrated edge, and tend to penetrate in depth, destroying the bone and cartilage. In the chronic form of the disease, which may last a long time,—months and even years,—the symptoms and signs in the nose and other organs are far less pronounced. Here a recovery is possible; but, as often happens, it becomes acute, and then, as a rule, it ends fatally. Acute "glanders" nearly always terminates in death by intercurrent pyaemia and septicaemia.

Malleus begins in some cases, according to *Babes*, with a chronic dry rhinitis. Then indurated infiltrations are formed, which, however, show little tendency to break down, yet here the bacilli lie dormant for a long time, until suddenly, subsequently to any trauma or acute disease, they are liberated to begin their deadly work.

Diagnosis.—Diagnosis is difficult in all cases where anamnesis is uncertain, for the changes in the nose have nothing peculiar, and, especially in the chronic form, may easily be mistaken for syphilis. The diagnosis, therefore, can only be made after a lengthier observation or, ex juvantibus, the exhibition of iodide of potassium. A bacteriological examination is sometimes very useful.

Treatment.—This is purely symptomatic. Cleansing the nose with disinfectants is the chief local treatment. Of more importance is the prophylaxis, which we need not here emphasize.

9. LEPROSY (LEPRA; ELEPHANTIASIS GRAECORUM).

The nose, according to *Sticker*, must be considered as the gate of entrance for the Bacillus leprae; an opinion, however, which has encountered much opposition. In many cases the external, as well as the internal, nose is mostly affected by leprosy; and in the tuberous variety, always (*Bergengrün*).

The general health is frequently disturbed. The nose is at first inflamed and swollen, but later on it is more dry and shows circumscribed or diffuse infiltration. These infiltrations are mostly seated at the vestibule and may undergo atrophy; but more often they break down after a time and form ulcers, which, like syphilitic ulceration, destroy all the tissues. There is a discharge of a peculiar, sweet, unpleasant smell, which forms scabs. In the later stages scars are formed which tend to contract, and so to lead to constrictions and adhesions. The skin of the nose and that of the neighbouring parts of the face and lips is chiefly infiltrated (leontiasis), or the nose may appear flattened, like that of a pig's snout. The affected parts are often insensitive, the insensibility also spreading to other parts of apparently healthy skin or mucous membranes.

Diagnosis.—Diagnosis, if the disease is of some duration, is not difficult, especially if the whole body is examined. In time the lepra bacillus can be found.

Prognosis.—Prognosis is, quoad vitam, unfavourable, though the disease is very slow and chronic.

Treatment.—This must be symptomatic. We can only cleanse the nose, and prevent, or try to prevent, adhesions or to separate synechiae.

VIII. FOREIGN BODIES AND PARASITES.

I. FOREIGN BODIES.

Etiology.—Foreign bodies gain access to the nasal cavity in various ways, most commonly through the nostrils, less often from the posterior nares, and rarely through accidental gaps in the nasal walls. Children, or at times insane persons, put all sorts of things into their noses—peas, beans, stones, buttons, coins, beads, paper, etc. In adults, one sometimes finds pieces of cotton-wool, gauze, or plugs, which had been left there and forgotten. Sometimes, from speaking whilst eating, or from swallowing "the wrong way" (dysphagia), or vomiting, particles of food may reach the nose through the choanae; or by injury; broken blades, splinters, bullets, etc., may penetrate into the nose.

Pathology and Symptoms.—Small, slippery, smooth, mobile foreign bodies are often removed by the act of sneezing alone. Larger ones, especially if the passages of the nose are narrow, remain most commonly in the lower meatus, where they rest and cause no other symptoms save slight atrophy through pressure. In time, however, they cause inflammation; the nose, on the side in which is the foreign body, becomes obstructed, and a discharge, at first thin, later more purulent and evil-smelling, appears. Associated with this there may be headache and other nervous symptoms, and in children even convulsions or delirium. The neighbouring cavities or organs may be affected (the lachrymal apparatus, the eye, ear, or antrum). If the foreign body is not then removed, erosions at the anterior nares or on the upper lip, or granulations and ulcers, may be formed, resulting in necrosis and destruction of the mucous membrane and cartilage. On the other hand, deposits of chalk and magnesia are formed round the foreign bodies, which are then converted into nasal calculi (rhinoliths).

In some cases the foreign body in the center of the rhinolith was overlooked; and in others, dried secretions or blood coagula have been found; masses also of leptothrix, were observed. The salts are derived from the naso-lachrymal secretions.

Nasal calculi may grow to a very large size. *Zuckerkandl* describes one 5 cm. in length and 2.5 cm. in breadth. Their shape varies according to the shape of the nasal cavity; their surface may be smooth, or uneven or spinous; and they show a great variety of colours. In cement-workers, a special kind of calculus has been found.

It may be mentioned that aberrant teeth have been found as the cause of "stone in the nose" (and in the antrum). This can only be explained by a congenital inversion of the dental germ.

Diagnosis.—The diagnosis is made by rhinoscopic examination, and with the aid of a probe; for the patient may not know that a foreign body has reached his nose, or has forgotten the fact as to when and how it came to be there. Children very often, from fear of punishment, do not tell the truth; and it is just in children that we meet with the greatest difficulty in making our diagnosis.

A one-sided, evil-smelling discharge from the nose in children points mostly to the presence of a foreign body. Usually it is the right nasal half which is affected, and is explained by the right-handedness of the children. Obstinate children must be kept under proper control, in order to examine and remove the foreign body; narcosis, however, will seldom be required for the purpose. The resultant blocking by secretions must be removed by cautious syringing, and we must examine as speedily as possible. In adults we can usefully employ cocaine-adrenalin.

The distinction between a calculus or necrosed bone (sequestrum), or a soft foreign body and a decomposing tumour is sometimes not very easy. Inflammation of the accessory cavities can seldom be misconstrued. In certain cases the x-rays must be resorted to.

Treatment.—The removal of foreign bodies from the nose must be done instrumentally. Other methods, as pressing in air by means of a *Politzer* bag, or syringing the healthy side, are uncertain, and are liable to cause inflammation of the middle ear. Whenever it is possible, before attempting removal, one should get information about the size, seat, and nature of the foreign body, resorting eventually to local anaesthesia with cocaine and adrenalin. It is best to introduce a strong probe, bent at the end, push it behind the foreign body, and strive to lever it forward, and so out of the nostril. Children must be firmly held by an assistant or some one else who takes the small patient upon his lap and keeps the legs between his knees, presses the head against his own forehead or chest, and at the same time prevents struggling by holding him firmly in his arms (Fig. 45).

In case of need, an anaesthetic must be employed. Pieces of paper or cotton-wool can be seized under guidance of a speculum with the forceps, and impacted bodies grasped with the *Heymann's* cupped forceps, and extracted. In some cases, as in large rhinoliths, extraction is only possible after crushing the stone. It is hardly permissible to push the foreign body backwards into the naso-pharyngeal space, but if there is no other possibility of removal, one must be careful to prevent the foreign body from slipping into the larynx by inserting two fingers into the post-nasal cavity through the mouth.

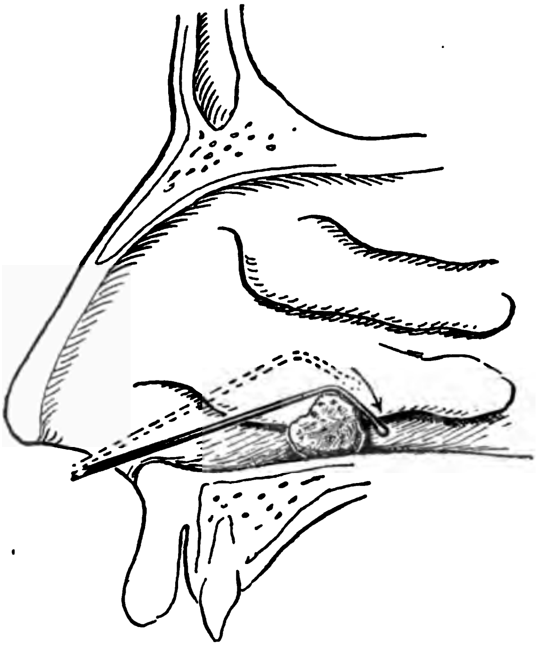


Fig. 45.—Removal of a foreign body from the lower meatus.

Haemorrhage after extraction is very insignificant, and any suppuration which previously existed soon ceases of itself. If it continues for more than a few days, we must suspect that part of the foreign body has remained behind, or that a second one is present.

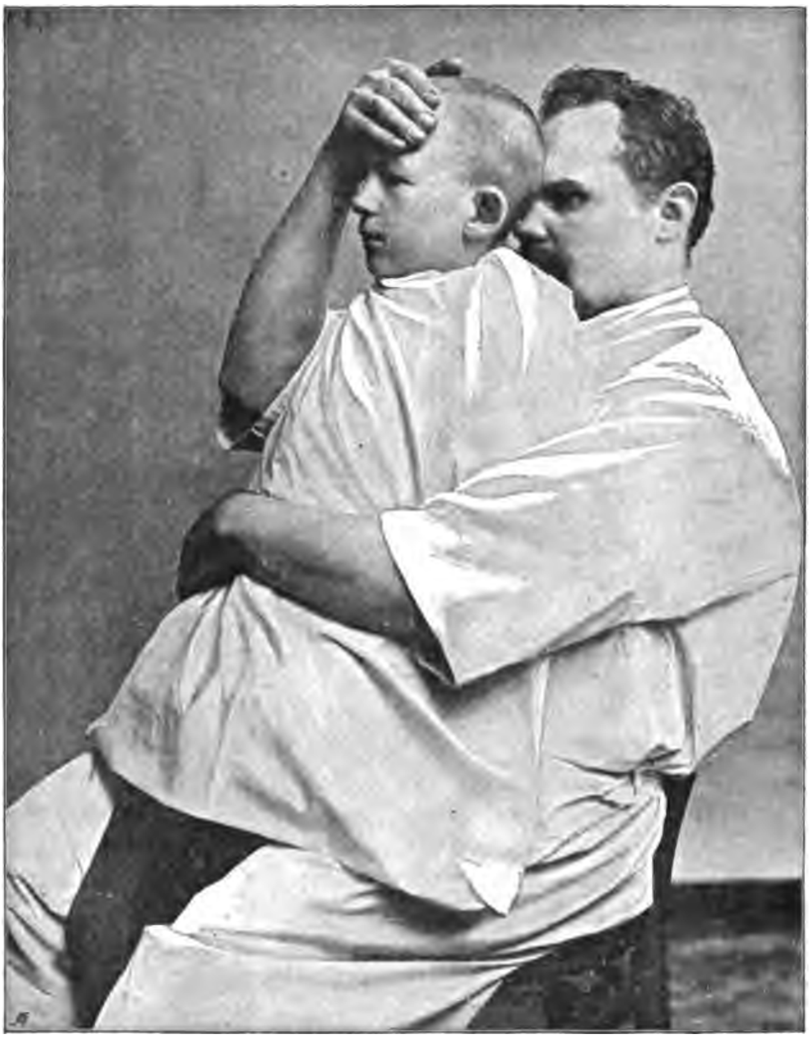


Fig. 46.—Holding the child during removal of a foreign body from the nose.

2. PARASITES.

Animal parasites are not often observed in our country, but more frequently in the tropics. There, the larvae of some kinds of flies or other insects are found, which, attracted by the smell of the secretions, lodge their eggs within the nose (myiasis), and into the nose of persons who sleep in the open air; or it consists of larger animals, of the class of *Scolopendrum* (centipede), ascaris, hirundo (leech), which perchance may have crawled into the nose.

Symptoms.—The symptoms are those of irritation, sneezing, discharge, headache, fever, etc. In myiasis ulceration may occur, with destruction of the nose, and even death from meningitis has followed.

Diagnosis.—Diagnosis is based on the finding of parasites. The larvae may be found in the discharge or in the nose itself, where they can be recognized by their movements.

Treatment.—Syringing the nose with diluted chloroform (chloroform and distilled water equal parts) or extraction of the parasites by means of instruments. Inhalations of vapours of a mixture of menthol and chloroform (chloroform, 5.0; menthol, 0.5) have been recommended in order to stupefy the animals and thereafter to remove them from the dilated passages. *Roorda Smit* recommends, as a sure and quick remedy in myiasis, the insufflation of calomel into the nose.

Vegetable, more rarely than animal, parasites are found in the nose. The Oïdium albicans (thrush), which has been observed, has probably spread from the mouth into the nasal cavity, but it is only found in unclean children who are very seriously ill. Some species of Aspergillus glaucus and Mucedo have also been found in the nose.

In such a case friable white or gray or darker patches are seen; they are, with the exception of the thrush fungus, clinically of no significance; the nature of these patches is easily recognized under the microscope (p. 213).

Treatment.—This consists in loosening the patches by probe, removing larger conglomerations by forceps, and in painting the affected parts of the mucous membrane with borax or chinolin (sodii biborat., 5.0, ad glycerin, 25.0); (chinolin, 0.4; glycerin and spiritus vini, āā 10.0).

ACTINOMYCOSIS.

The germ of this disease, which may commonly be seen on the ears of corn as the fungus, actinomyces, usually enters the body through the mouth (carious teeth, through the gums, or lacunae of the tonsils). In the nose, actinomycosis is very rare. In a case described by *de Simoni*, the nasal cavity was filled with a reddish, fleshy, and readily bleeding mass, which was adherent to the middle and lower turbinal and the floor of the cavity, and bulged the cheek and palate forwards. In the middle of the hard palate a small ulcer was noticed, through which a probe could be pushed into the nasal cavity.

Diagnosis.—As regards diagnosis, tuberculosis, syphilis, and malignant new-growth must be considered. The diagnosis is ensured by the microscope; when one will find the characteristic conglomeration of threads and clubs formed by the thickened ends of the mycelium.

Treatment.—This consists in scraping off the fungoid patches, opening the abscesses, and giving internally large doses of iodide of potassium.

IX. TUMOURS.

1. BENIGN NEW-GROWTHS.

(a) MUCOUS POLYPI.

Etiology.—Of all the new-growths occurring in the nasal cavity, the mucous polypus, or, as usually designated, nasal polypus, is the most common. They occur oftener in men than in women, and seldom in children. The polypi, as well as the polypoid hypertrophies before mentioned (see p. 77), are most probably always the result of chronic inflammation, and we are often able to see both present together, at the same time, in chronic nasal catarrh, the one merging into the other. Chronic suppuration or a foreign body very often gives rise to polypoid growths; and sometimes a malignant tumour may be the cause, or no discharge at all may even be noticed, but in such cases, however, the etiology is unknown. We do not know, also, why, in one case of chronic inflammation, polypi are formed, and in another, no such thing happens.

Pathology.—Nasal polypi are of a gray, gray-yellow, or grayish-red, glazed colour. They are soft and jelly-like in consistence, and are freely movable, as they mostly possess a pedicle. They almost always arise from the middle concha (Fig. 37, p. 77), or the edge of the hiatus semilunaris, more rarely from the roof; and in some unique cases, from other parts of the nasal cavity. Not infrequently, however, they come from the accessory cavities, and in particular from the ethmoidal cells.

At first the polypi have a globular or conical shape; but later on their growth is regulated by the configuration of the nasal cavity. If the lumen be wide, the pear-shaped form prevails; and in a narrow lumen the polypi are more flat, like a bag or cockscomb. There is seldom only a solitary polyp; mostly, there are several, often dozens of them, though only a few large-sized ones may be seen at the first examination. Their mobility depends on the length and thickness of their pedicle, which may be round, like a string, or broad, like a band; and sometimes several polypi depend from one stalk. At times they grow backwards into the post-nasal cavity. In the majority of cases they occur in both sides.

Nasal polypi appear as oedematous, degenerated, circumscribed, hypertrophic outgrowths of the nasal mucous membrane, covered in some places with ciliated epithelium, at other places with stratified epithelial cells. They consist of a loose connective-tissue network, imbued with oedema, and which, in some cases, shows cystic degeneration or even real cysts, derived from dilated mucous glands. In rare cases the glandular tissue preponderates, and thus true adenomata (fibroadenomata) are found. The oedematous fluid, which we must consider as the product of a passive serous transudation, contains large quantities of albumin, but no mucin, and hence the usual name, "mucous polypus," is a misnomer.

The pedicle contains many blood-vessels, and that is the reason why we meet with severe bleeding if we cut through the polypi at their base, whereas there is hardly any bleeding if we cut through them more peripherally.

Symptoms.—Small polypi do not give much cause for complaint. There is irritation in the nose, causing frequent sneezing, a discharge of thin, watery, clear secretion, or a slight frontal headache—all symptoms which many persons can put up with uncomplainingly. In larger sized polypi, however, respiration through the nose becomes hampered, with all its consequences (headache, giddiness, depression, irritability, dryness in the throat, nasal speech, loss of taste and smell, etc.). Secretion is increased, and the patients themselves have the sensation of a body floating to and fro during respiration. For the connection between nasal polypi and asthma see p. 128.

Diagnosis.—Abnormally large polypi appear, in neglected cases, at the nostrils, where they can be seen without any difficulty; otherwise rhinoscopy, either from the front or back, and probing will afford us every information we desire with regard to size, consistency, colour, and mobility. Difficulties may arise, if the nose is narrow, or if there are protrusions or deviations of the septum. In such cases we are much aided by painting with adrenalin. We must also ascertain whether we have to deal with simple uncomplicated polypi or whether they are only secondary to a primary disease (suppuration of an accessory cavity, foreign body, malignant growth, etc.). Purulent secretions or even evil-smelling secretion flowing between the polypi over the conchae; or if we touch on rough bone; or should the polypi bleed on gentle touch with a probe—should all cause us to suspect complications.

We should make it a matter of routine to examine both sides, although the report of the patient may point to one side only.

Prognosis.—As far as relief or cure of the complaint is considered, prognosis is good; but it is somewhat apt to be disturbed by the liability of the polypi to recur, even though we may have removed them and destroyed their site of origin completely. If, on the other hand, we can remove the cause (chronic catarrh, catarrh of antrum, etc.), a radical cure may be established.

Treatment.—The best means of removing nasal polypi is by the cold snare. Having anaesthetized the mucous membrane, *Krause's* snare is introduced in the same way as we have described for the treatment of polypoid hypertrophies (p. 80).

One must keep close along the septum, and only in exceptional cases along the lateral wall; then push the loop gently upwards and over the first polypus met with, or, if possible, over several, and as far up as their seat of origin. Then the loop is constricted, and with a short jerk the polypus, together with its stalk, is torn off; and is often accompanied by the matrix. (See Fig. 47.) In any case, the method of tearing is more preferable and more radical than that of cutting, which is much recommended. The operation is painful, but nevertheless so quick that we may well risk the pain and eventual possible haemorrhage. In certain cases, especially where the introduction

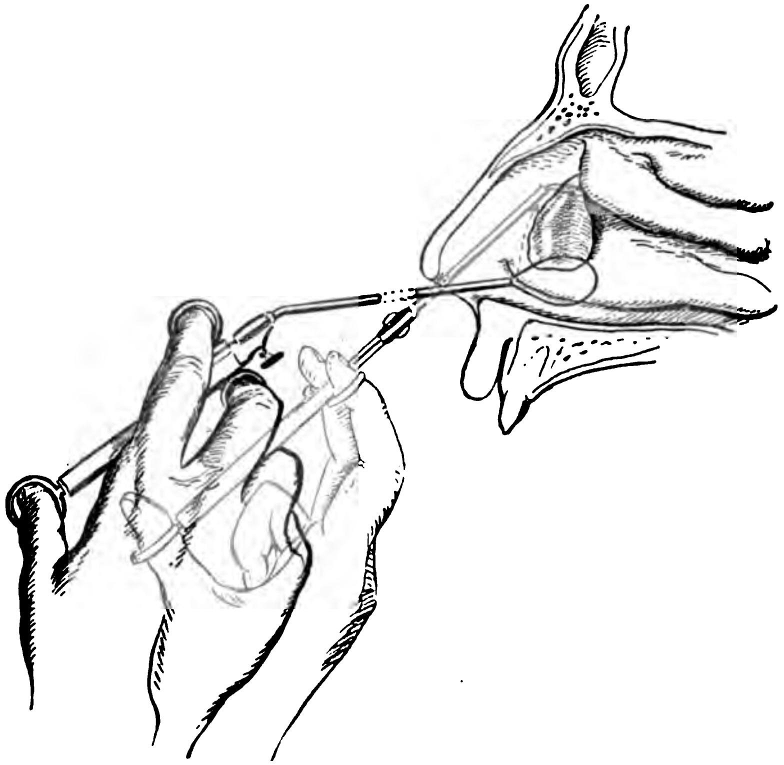


Fig. 47.—Removing a nasal polypus. The black drawing shows the introduction of the snare, with the loop in the vertical position (plane). The red drawing shows the turning of the loop into the horizontal plane; the pushing upwards to the seat of origin; and the constriction of the loop.

of the snare meets with great difficulty or the polypus offers much resistance to the pulling off, we may take refuge in the cutting through process. The smaller the polypi and the higher up they are seated, the more difficult is it to fit on the wire loop. In such cases, we may sometimes succeed by using very thin wire to form the loop, and by making it as small as possible.

Smaller polypi and the eventual remains of larger ones can afterwards be nipped off with *Heymann's* forceps. It often happens that the operation must be interrupted on account of the haemorrhage, or several sittings are required. In such cases it is a good plan to make short interruptions and to alternate the sides, operating now on the one, then on the other, side, and so on. One should also, for tactical reasons, remove at the first sitting as much as possible, in order to relieve at least one side of the previously obstructed nose. If the ethmoidal bone is found implicated, the ethmoidal cells must be opened, and eventually parts of or the entire middle turbinate bone must be resected or amputated. (See the chapter on the Diseases of the Ethmoidal Labyrinth.)

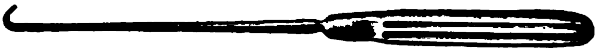


Fig. 48.—Lange's choanal hook.

Special difficulties arise at the removal of polypi growing into the naso-pharyngeal space. Here we often fail to fit the loop. In such a case the choanal hook of *Lange* (see Fig. 48) can be used with advantage. This is introduced, with the point directed downwards, close up to and along the septum; the point is then turned laterally, so that it engages round the pedicle of the polypus, and it is then pulled, in short jerking movements. With the finger of the other hand we can suitably guide the hook from behind.

In the more tough or broad-seated polypi we can use the hot (galvano-caustic) snare with greater advantage. After removal, it is very important to destroy the matrix by cauterization with the galvano-cautery, chromic or trichloracetic acid, in order to prevent recurrence—unfortunately, however, not always with success. In some cases we receive the impression that on account of too energetic treatment, the growth of the polypus has been stimulated.

BENIGN TUMOURS OF SPECIAL KIND.

What we have said in previous chapters concerning the polypoid growths on the conchae, and in particular on the lower one, it is not here necessary to repeat, and we, therefore, refer the reader to Chronic Rhinitis (see page 76). The hypertrophic growths there described, which have their favourite seat on the lower concha, are variously named by different authors. They are called soft papilloma, if the surface is smooth and lobulated; or fibro-angeioma or angeioma, if they show great vascularity and are consequently more red and fleshy in appearance.

The bleeding polypus of the septum, which also shows intense vascularity, is not, in its appearance, distinguishable from a vascular hypertrophic polypoid growth. It appears as a growth of the size of a pea or walnut, with smooth or lobulated surface, and is nearly always broad based upon the anterior part of the nasal septum, where it gives rise to obstruction and bleeding. It is said to occur more frequently on the left side than on the right; and oftener in women than in men.

From the soft papilloma the true hard papilloma (fibroma) must be distinguished; the latter occurs as a warty outgrowth at the entrance of the nose or septum, seldom in other parts of the nose. It consists of a network of fibrous connective tissue interspersed with epithelial cells.

Occasionally, lipoma, chondroma, and osteoma have been observed as occurring in the nasal cavity.

Treatment.—Treatment consists in removal of the tumours, either by means of the hot or cold snare. The chondroma and osteoma, however, are mostly too large to admit of an operation solely through the nose.

2. MALIGNANT TUMOURS.

Malignant growths in the nasal cavity are, on the whole, not often found. Relatively, the most common is sarcoma; less frequently, carcinoma, and very rarely, lympho-sarcoma, which probably always spreads from the naso-pharynx into the nasal cavity. Usually, the tumour comes under our notice if it has produced marked clinical symptoms, *i. e.*, at a time when it has already grown so large as to fill the nose. It is then often impossible to say what is the primary seat of the tumour, whether it originated in the nose primarily or grew into it secondarily from the vicinity. These tumours, and in particular sarcoma, take their origin mostly from the upper parts of the nose, especially from the upper part of the septum.

Sarcoma occurs mostly in youth and middle age; carcinoma, mostly in elderly people.

Symptoms.—Symptoms consist in obstruction of the nose, pain, neuralgia, nose-bleeding, and later, if the new-growth increases in size, of disturbances of the auditory apparatus (difficulties of hearing, tinnitus in the ear, etc.), or it may invade the orbit and cause displacement of the eye, exophthalmos, paralysis of the ocular muscles, even amblyopia or blindness. The tumour often ulcerates, and there is then a foul-smelling discharge from the nose. Carcinoma has a greater tendency to ulceration than sarcoma.

Externally, we at once notice a broadening of the nose, and exophthalmos, or bulging of the cheek or the hard palate, may be depressed as a consequence.

On rhinoscopical examination one finds large broadly seated masses with a lumpy or warty surface, and free bleeding on touch. Mucous polypi can often be seen in the neighbourhood, and in such numbers that they may conceal the tumour to the inspecting eye. The surface of the tumour shows ulceration and is coated with a slimy, foul discharge.

Diagnosis.—The diagnosis of malignant tumour is not always easy at first. The surgeon must beware of mistaking it for syphilitic or tubercular processes. Microscopical examination is likely to decide any doubt, but it must be borne in mind that round-celled sarcoma shows microscopically the same structure as a gumma. In such a case a trial with iodide of potassium would be justified. Later on the rapid growth, the spreading into all the adjacent tissues, and the affection of the general health will leave no doubt as to its malignant nature.

Prognosis.—Provided that the cause is not already too far advanced and is discovered in good time, sarcoma might give a better chance of cure than carcinoma; both are, of course, unfavourable.

Treatment.—If we have the opportunity of seeing the tumour at its commencement, we must, by all means, endeavour to destroy or remove it, even with the healthy tissues in its neighbourhood. The galvano-cautery or the hot snare is best used for this purpose; electrolysis is also spoken of favourably. Internally, arsenic (in pills or Fowler's solution) may be given.

But if the new-growth be of larger size or be seated high up in the nasal cavity, a radical operation is indispensable (resection of part or the entire nose or of the upper maxillary bone, etc.). It is often too late for operation, and then we are compelled to limit our help to palliative treatment (douching, local anaesthetics, narcotics, attention to general health, etc.).

X. NERVOUS LESIONS.

1. DISORDERS OF THE SENSE OF SMELL.

The sense of smell may be diminished, completely lost, pathologically increased, or otherwise altered. Usually, the pathological alteration of smell is only symptomatic of another primary disease of the nose or its accessory cavities, but it may appear as a genuine disease, a morbus sui generis.

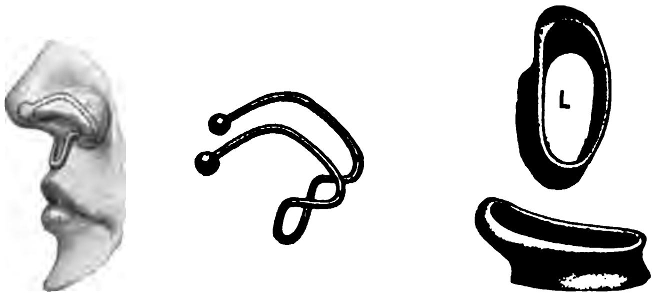
(a) Hyposmia and Anosmia.

Etiology.—Diminution (hyposmia) and loss of smell (anosmia) are due to—(1) *respiratory,* (2) *local,* (3) *central,* causes.

(1) Respiratory hyposmia is produced by the odourous substances not being allowed to reach the regio olfactoria, as in synechiae, adhesions, swelling of the mucous membrane in catarrh, deviation of the septum, tumours, and adhesions of the alae nasi. (2) Local causes of hyposmia are diseases of the regio olfactoria and of the mucous membrane, such as atrophy of the pigment of the olfactory cells, due to poisoning (cocaine, morphine, nicotine, atropine); or are due to douching with certain strong fluids or repeated exposure to noxious vapours

(trade anosmia). Another class of causes are peripheral neuritis (as in influenza) and degeneration (atrophy in chronic rhinitis). Sometimes anosmia is of traumatic origin. (3) Central hyposmia and anosmia—usually the latter—is caused by disease of the olfactory nerve (itself or of its cerebral center of origin). To this class belong the frequently unilateral anosmia hysterica, the anosmia due to traumatic neurosis (traumatic hysteria), climacteric and congenital anosmia.

Symptoms.—Loss of smell is either complete, or is only absent for certain substances (partial anosmia), or there exists parosmia. Anosmia, and especially hyposmia, is often not apparent to the patient himself. Rhinoscopically, we find nothing save in respiratory or atrophic hyposmia.



a *b*

Fig. 49.—Nasal elevator: (*a*) *Feldbausch-Roth;* (*b*) *Schmidthuisen.*

Diagnosis.—We examine the power of smell by the method before described (see p. 16). Distinctions between the various forms of anosmia can only be made if we have a sufficient history of the case, from the resulting objective examination, and last, but not least, per exclusionem.

Prognosis.—Prognosis is not always bad, and depends on the primary cause; it is best in respiratory and worse in central and long-standing cases.

Treatment.—Should aim at removing eventual causes (obstruction, catarrh, deviations, etc.). In order to relieve the discomfort occasioned by the nasal wing becoming pinched on forcible inspiration, *Feldbausch-Roth's* or *Schmidthuisen's* elevators are very useful (see Fig. 49). In syphilis we may give iodides, and in hysteria we apply the galvanic current two or three times weekly; one electrode is placed on the neck and the other in the nose. In some cases insufflation of strychnine (0.05 to 10 amylum) has a good effect, given twice or three times daily, snuffed up or insufflated.

(*b*) HYPEROSMIA.

If the acuteness of the sense of smell is intensified, the condition is spoken of as hyperosmia. As acuteness of smell varies within certain physiological limits, hyperosmia may be physiological.

Symptoms.—Its symptoms, however, are at times so marked that the sufferers feel themselves very inconvenienced by it and its consequences (headache, vomiting, giddiness, etc.). It is mostly in hysterical or neurasthenic persons or pregnant women that we meet with high degrees of hyperosmia, often accompanied by parosmia.

Treatment.—Bromides may be given internally, but of more importance is the general treatment (diet, rest-cure, and general hygiene).

Locally, painting with a weak solution of cocaine sometimes does good.

(*c*) PAROSMIA.

The condition is that of parosmia if certain scents are perceived in a wrong way—mostly unpleasant (subjective cacosmia)—or if persons perceive a smell where, de facto, there is no smell (olfactory hallucination).

Parosmia, like the previous hyperosmia, occurs mostly in hysterical or neurasthenic persons. But it has also been observed in influenza and acute coryza, as also in various mental disorders. It might sometimes occur as a symptom of suppuration in the antrum, foreign bodies or malignant growths in the nose, and is then called objective cacosmia.

Prognosis and Treatment.—As regards prognosis and treatment, there is not much to be said, for they depend on the primary causes.

2. DISORDERS OF SENSIBILITY.

The nasal mucous membrane is generally very sensitive, because of the abundant supply of nerves. All sorts of manipulations, even a slight touch of the probe, excites, if not always pain, anyway unpleasant sensation; nevertheless, the interior of the nose, especially the lower part, is remarkably tolerant of pathological processes. This is why we hardly ever observe pain after operative manipulation. It can now be easily understood why it is often difficult to say whether the sensibility of the mucous membrane is diminished or increased. Besides, it depends on so many individual qualities. It is, however, relatively simple to ascertain total insensibility.

(a) ANAESTHESIA.

If anaesthesia occurs alone and on one side only, it points almost certainly to hysteria, and is due to peripheral or central functional paresis of the fifth nerve. Loss of sensibility is not often noticed, probably for the reason that the patient has nothing of which to complain. On examination, one is surprised to find the mucous membrane insensitive. Anosmia, often reported by the patient, is in such a case due to the non-perception of such acrid substances (vinegar, sal ammoniac, etc.) as normally stimulate the trigeminal nerves. (See p. 15.)

Artificial anaesthesia can be produced by certain drugs (such as cocaine, alypin, etc.).

(b) HYPERAESTHESIA.

Increased sensibility may be so intensified as to amount to neuralgia, though the latter is rarely limited to the nose, and the pain in that case always radiates into the neighbouring organs, eyes, teeth, jaw, and forehead. Hyperaesthesia is a stigma of hysteria and neurasthenia, and is the cause of many reflex neuroses in those diseases.

(c) PARAESTHESIA.

Paraesthesia is far more frequently observed in the throat than in the nose. The patient complains of feeling hot or cold, or has a sensation of a foreign body, or of itching, in the nose.

It often happens that the patient does not know how to describe or locate the strange sensations. Anatomical changes are usually absent or so slight that the urgency of the symptoms complained of cannot be explained by them.

Treatment.—Treatment in all these cases of paraesthesia must be guided by the primary cause (hysteria, catarrh, etc.), and our chief attention should be directed to ameliorate the general health by proper diet and change of climate, arsenic, and last but not least, suggestion.

3. NASAL REFLEX NEUROSES.

Etiology and Pathology.—It is well known that, by stimulating certain organs, as, for instance, the uterus, intestines, sole of the foot, larynx, and auditory meatus, we are able to excite a reaction in other, sometimes quite distant, organs, which, within physiological limits, concerns chiefly the muscular, vasomotor, or secretory spheres. It is the same with the nose.

(*a*) As of first importance, we must enumerate sneezing and lachrymation. (*b*) Respiration and cardiac function are influenced by reflex action from the nose. (*c*) Lastly, the increase and decrease of swelling of the cavernous tissues in the nose are, after all, but reflexes.

In persons who, by hereditary predisposition, by overstrain, or by exhausting maladies, have become generally or locally hypersensitive (see previous chapter), the irritability of the nasal mucous membrane might be so much exaggerated that relatively insignificant stimuli are, indeed, sufficient to excite in the nose or other organs reflex symptoms of a muscular, vasomotor, or secretory nature. Certain other irritative symptoms in the sensory sphere (neuralgia of the trigeminal, headache, etc.), observed in chronic rhinitis, are not caused by pure reflex, but must be considered as concomitant manifestations or direct complications of the primary disease.

It is always well to examine carefully if an apparent disorder is not due to anatomical changes. For in our "nervous age" "neurosis" having become part and parcel of the most varied and every possible disease all the world over, much sails under the flag of "nasal reflex neurosis" which, to my mind, has to be separated from it entirely. Indeed, "nasal reflex neuroses" are much less common than some enthusiasts would have us believe at present.

Besides a psychopathic predisposition, a pathological reflex action requires an exciting cause. This can be most commonly found in mechanical irritation of the fifth nerve by polypi, catarrh, tumours, foreign bodies, touching, probing, dust, etc. Or the irritation might be caused by two opposing folds of mucous membrane (in catarrhal affection) rubbing against each other, or the cavernous bodies might easily swell and establish the contact, so to speak.

Stimulation might be chemical or thermal, or, finally, olfactory. In the latter case, the olfactory nerve forms the afferent path. We speak, in such a case, of idiosyncrasy for certain odours (flowers, especially roses, fruits, and drugs). A reflex may be excited, and this is generally accepted, from all parts of the mucous membrane, but most easily from certain irritable zones. (See p. 15.)

Symptoms.—The clinical symptoms of the nasal reflex neuroses vary according to the two ends of the reflex arc. Both stimulation and reaction may take place in the nasal cavity, or one may be in the nose and the other in a different, sometimes quite distant, organ.

We may, for practical purposes, divide the reflex neuroses into two groups: (1) Where both reflex and stimulation are in the nose; (2) where the one or the other affects a different organ.

INTRANASAL REFLEX NEUROSIS.

Nervous Cold.—The general practitioner who wishes to orientate himself in the difficult ground of reflex neurosis might well take nervous coryza as a paradigma for the whole class of similar disorders.

Symptoms.—Nervous coryza is characterized by the following symptoms: excessive swelling of the cavernous turbinal tissue, profuse discharge of a clear watery secretion, paroxysms of sneezing, and lachrymation. All these symptoms, however, are not always present at the same time, nor in the same degree. Sometimes one, sometimes another, may prevail, or one symptom alone may be so pronounced as to dominate the whole picture, and so form a variety of the disease. ["Hay-fever," which in its clinical appearance resembles reflex neurosis so much, cannot be considered as such, for *Dunbar* has shown that it is produced by a specific cause. We have, therefore, treated "hay-fever" as a separate disease. (See p. 90.)]

Nervous coryza may be distinguished from the ordinary inflammatory cold by its sudden onset; that it occurs in paroxysms; and sometimes quite regularly at a certain hour; lastly, that the secretion is always clear, never purulent.

The exciting stimuli are manifold. We mention the various scents, especially roses ("rose," "peach," and "stable" colds); particles of dust also, which have reached the nose may be considered, though it would here be difficult to draw the line between inflammatory catarrh and nervous coryza. Extranasal stimuli are: sudden cooling of certain parts of the skin (*i. e.*, through wetting the feet), sexual excitement, drinking quickly warm alcoholic liquors, indigestion, flashing of a bright light, etc. Relatively, the least trouble is caused by the congested intranasal cavernous tissue, which may only be a symptom by itself, but may also be the exciting cause of reflex action. It may, however, lead to permanent swelling of the concha. Very troublesome, indeed, are paroxysms of sneezing, which sometimes occur very frequently and follow each other rapidly; they may, by the bursting of a blood-vessel, provoke epistaxis.

One of the author's patients, when he left his warm bed every morning, was subject to 50 to 60 attacks of sneezing. A spina septi, which irritated the turgid concha, was discovered to be the cause. After removal, the attacks ceased permanently.

Very unpleasant, indeed, is the copious, sometimes excessive, watery secretion from the nose (hydrorrhoea nasalis) which is often associated with intense lachrymation or headache, roughness in the throat, migraine, and trigeminal neuralgia.

Erythema of the External Nose.—Here, as in the excessive swelling of the cavernous bodies, it is of the nature of a vasomotor reflex neurosis. (See p. 52.)

Influence of the Sexual Sphere on the Nose.—It is difficult to decide whether nose-bleeding, which occurs occasionally during or post coitum, or in abusus sexualis, masturbation, and coitus interruptus, is due to reflex or to the bursting of small vessels from increased blood-pressure. Vicarious nose-bleeding might be explained somewhat as a reflex action. The relations between the female sexual organs and nose are illustrated by the fact that intranasal manipulation during the time of the period causes more profuse haemorrhage. This is why we should operate only in urgent cases during the time of menstruation. According to *Kuttner's* and the author's observations, menstruation, in the majority of cases, during its course, continues unaccompanied by any subjective or objective change in the nose.

REFLEX NEUROSES IN OTHER ORGANS.

Asthma.—Bronchial asthma is a real reflex neurosis, which is brought on, according to *Fränkel,* by three different processes, viz.: (1) Spasm of the diaphragm; (2) spasm of the bronchial muscles; (3) catarrhal infiltration of the lung. The efferent neurons run, therefore, in the phrenic, vagus, and sympathetic nerves. The peripheral locus excitationis lies in various places, not the least common being in the nose. In such a case the asthma is spoken of as "nasal asthma," or, as *Eichhorst* terms it, "asthma bronchiale ex naso." Local irritations which tend to produce "nasal asthma" are of the same category as those mentioned in the previous chapter. Certain pathological changes in the nose, however, such as turgid conchae, deviations of the septum, adhesions or atresiae, especially polypi of the mucous membrane and foreign bodies, are supposed to be the malificent agents, which, by pressure or other irritation, cause the asthma. It is as well not to jump to a conclusion too quickly as to the real connection of these circumstances with asthma, for one finds that, among the great number of patients suffering from nasal polypi and other diseases, there are only a very few in whom an asthmatic attack can be artificially provoked, on the occasions of either diagnostic or therapeutic intranasal manipulation. But it should also be borne in mind that an original "nasal asthma" after a longer or shorter period of time may change its characters, so that, finally, all possible varieties of extraneous irritations may provoke an attack.

There is no solitary particular spot, but many places in the nose from which an asthmatic attack might be excited.

1. The asthmatic attack often begins with an aura, *e. g.*, some (general) forebodings or warnings known to the patient, and in other cases it is preceded by a prodromal stage of tickling, paroxysms of sneezing, or nasal hydrorrhoea.

2. *Spasmodic Cough.*—Mechanical irritation of the nasal mucous membrane is apt to cause in some nervous persons paroxysms of coughing. This nasal or trigeminal cough is distinguished by its lack of any expectoration; the larynx, throat, and trachea being perfectly free of the same. This nervous cough can be the more easily provoked by touching the posterior wall of the pharynx with a probe.

3. *Cardiac Neuroses.*—In these, just as under normal circumstances, the heart can be still easier influenced from the nose in a neuropathic individual. One thus observes palpitation, oppression and apprehensions, pain or zona, etc., but it is, however, not at all certain whether these symptoms are always reflex, or sometimes functional.

4. *Dysmenorrhoea.*—The fact, first reported by *Fliess*, that dysmenorrhoea can be beneficially influenced from the nose, has been asserted by many authors, though not in so large a measure as maintained by *Fliess*. Dysmenorrhoeic pain in the back often ceases, if the tubercula septi, and uterine colic if the lower conchae, are painted with cocaine; and it has been reported that they cease permanently if these "genital spots" are cauterized with trichloracetic acid or the galvano-cautery. It has been found that these results can also be achieved by the employment of quite different means. Some authors consequently deny the specific effect and ascribe it to suggestion.

5. *Other Neuroses.*—In some unique cases—whether right or not, we do not desire to assert—spasms of the glottis, of the face ("tic convulsif"), migraine, epilepsy and hysterical fits, and salivation, have been described as being capable of being excited or influenced from the "spots" in the nose.

Diagnosis.—It is always difficult to prove that any neurosis in question has its origin in the nose; and we should never—within certain limits—be satisfied if we do not succeed in exciting an attack by touching the nasal interior with a probe, and then to stop the same by painting the particular spot with cocaine. The painting is done by sponging the mucous membrane with a piece of cotton-wool soaked in a 15 per cent. solution of cocaine, or by spraying the nose with a 10 per cent. solution.

The positive effect of cocainization is not always an absolute proof that the nervous disorder is dependent on a lesion in the nose; for it is impossible to exclude the element of "suggestion." On the other hand, also, a positive experiment with the probe is not an absolute test, because in hysterical persons these attacks can be simulated and easily excited (*Kuttner*). The negative result, equally, of course, in both experiments, proves nothing. Still less reliable are the results of treatment, as success is often due to "suggestion" or must be ascribed to the inhibitory effect of counterirritation.

Diagnosis is aided here by anamnesis (see p. 31); by the negative result of examination of the other organs, and the concurrence of other various reflex disorders. Nasal disease is not in itself a proof that the neurosis is of a nasal character. If all methods of examination have failed, and other causes cannot be found, then we are justified, having discovered a nasal disease, in making a diagnosis of probable nasal reflex neurosis.

Prognosis.—Prognosis is best in those cases where we find pathological changes in the nasal cavity, so that, by treating or removing them, we can cure the reflex neurosis. Indeed, in some cases we are able, by an operation, to definitely cure. But in long-standing complicated cases the chances of establishing a lasting cure are very small, and in very nervous patients prognosis for those reasons is bad.

Treatment.—Treatment must be general and local. Locally, we try to exclude the ends of the afferent neuron. This can be done by destroying them chemically or by galvanocauterization, or by removing the exciting abnormalities, *e. g.*, polypi, synechiae, adhesions, etc., by the methods before described (p. 55, et seq.). How much of the therapeutic success must be attributed to "suggestion" we do not care to say. In some cases the effect of operation appears much later, is sometimes missed altogether, or has just the opposite effect to that which we expected, namely, by producing just the reflex action we desired to cure. We must, therefore, not forget that nervous patients react differently after operation, and, therefore, we must not be too rash or hasty in our local treatment. It is in these not too clear cases pathologically, under the idea that an intranasal operation would act as an effective suggestion, that one may be induced to perform all sorts of superfluous or even senseless manipulations, and in whom the galvano-cautery, which has ever made a deep impression on the patient, is used more often than is necessary or beneficial. We certainly do not deny that galvano-cauterization may be useful, even if it does sometimes seem superfluous, yet we should not allow ourselves to be too optimistic as to its success.

In bronchial asthma long-continued treatment can only be beneficial if free respiration through the nose is established. Here we must take great pains to find any particular spots from which the reflexes can be excited.

The general treatment is, perhaps, the most important of all in these kinds of disorders, and we need not discuss it here, for it is contained in the text-books on Internal Medicine or Nervous Diseases.

THE DISEASES OF THE ACCESSORY CAVITIES.

The diseases of the accessory cavities are, on the whole, the same as those of the main cavity, but they are distinguished by certain peculiarities which find their explanation in the anatomical conditions, especially in the position and shape of their openings and in their relation to important structures, *i. e.*, eye, brain, ear, teeth, etc. Of the greatest interest to us in regard to the above are the inflammatory affections, which are now better recognized because of the improvement in their diagnosis.

I. INFLAMMATIONS.

(*a*) GENERAL.

Etiology and Pathology.—In the etiology of the various inflammatory processes of the accessory cavities, infectious diseases, and especially influenza and acute coryza,—the infectious nature of which can be no longer doubted,—pneumonia and scarlet fever in childhood, take the foremost rank (*Preysinq, Lange*). The only question is, how do the infectious germs reach the accessory cavities? Possibly the inflammation of the antral mucous membrane is produced by extension from the nasal mucous membrane as the result of continuity. Equally probable is it that infection of the accessory cavities has taken place at the same time as that of the main nasal cavity, just as we likewise see it in disease of the middle ear and mastoid antrum. Here and there, then, the disease, from its commencement, would be due to the virulence of the micro-organisms or to unfavourable, general or local, somatic conditions.

Other causes are: Injuries, foreign bodies, parasites, new-growths, gummatous processes, which spread from the nose into the wall of the cavities, and alveolar periostitis (carious teeth). Sometimes violent blowing of the nose, whereby infectious matter may be forced into the antrum, may be the origin of the disease.

Suppuration (inflammation) of an accessory cavity becomes chronic if the discharge of the secretion is obstructed or impeded, viz., through the secretions becoming thick, the openings being narrow or occluded, deformities of the nasal cavity (septum), polypi, or hypertrophies being formed in the neighbourhood of the ostia, in consequence of the constant irritation of the mucous membrane.

In long-standing suppuration of an accessory cavity the middle concha shows hypertrophic swelling and polypoid growth; later, on account of the rodent effect of the purulent secretion, the inferior concha also becomes involved and atrophies. In time the atrophic process spreads further and further, implicating the previously hypertrophied structures as well, until, finally, the nose shows atrophic rhinitis, with or without foetor. In this case the ozaena is certainly secondary to a primary suppuration of an accessory cavity. But there are many authors who maintain that the suppuration of the accessory cavity would be secondary to a primary ozaena. Repeated acute attacks of catarrh predispose the suppuration to become chronic. Sometimes, however, suppuration, even though it might have lasted for a long time, does not become chronic. In such a case the secretion of the antrum dries up; or becomes changed into a cheesy matter, and so forms a kind of foreign body, which quickly disappears after thorough washing out.

Pathological Anatomy.—In acute inflammation the mucous membrane is more or less reddened, swollen, and in some cases so much so that it is separated from the wall of the cavity, as in chemosis of the conjunctiva. Sometimes, however, there is no exudation; in others, again, the secretion is serous, mucous, muco-purulent, or purulent, rarely fibrinous; and cystic degeneration has also been observed.

The chronic inflammatory process shows two stages: (1) at first, the mucous membrane is oedematous; (2) later on it assumes a more fibrous character, with villous or warty excrescences thereon. The mucous glands soon degenerate and form cysts. The bone becomes implicated, is thickened, and osteophytes (exostoses) are formed, making the surface spinous or tuberous. The exudation is not often serous, but mostly purulent, and becomes thick and foetid if it has the opportunity to decompose. The bone may be eaten away (caries), but this latter is denied by some authors, or becomes atrophied, bulged, and thinned out, like parchment, so that on probing it shows the well-known symptoms of "parchment crackling" (sinusitis cum dilatatione). If the ostium is occluded for a longer time, a mucocele may be formed if the secretion has been mucous; or an empyema (pyocele) in purulent exudation. The cavity is frequently dilated through the formation of cysts or polypi.

The objective signs are determined by the secretion and complication of other organs. We have already, from a general point of view, discussed this matter (see Chapter IV., p. 75, et seq.) Here we shall treat the details in accordance with the special condition of each accessory cavity.

Diagnosis.—The diagnosis of the inflammation of the accessory cavities is not easy, especially if we have before us what is called a latent empyema. The expression between latent and manifest empyema is not happily chosen; for, strictly speaking, latent empyema can only be a suppuration in a closed cavity, making no subjective or objective symptoms whatever, and is only discovered by mere chance on examination. But in clinical jargon, the term has been accepted in the sense that it signifies a suppuration in an accessory cavity, without causing much trouble.

As has already been mentioned, the subjective symptoms can be very little relied on and the objective signs are extremely variable.

If the patient complains of headache, especially on one side, and in the forehead, over the root of the nose, and if these headaches are paroxysmal or periodical, accompanied by lachrymation and considerable purulent discharge from the nose, and perhaps also cacosmia, then the probability is great that we have to deal with suppuration in an accessory cavity. In acute cases the anamnesis will strengthen our suspicion. Examination of the patient is conducted by anterior, and then followed by posterior and medial, rhinoscopy. (See p. 16.) Any copious collection of pus must be cleared away by douching and sponging the nasal cavity. If, soon after the cleaning, fresh pus appears in the upper parts of the nose, in particular in the region of the hiatus semilunaris, and if the pus, moreover, shows pulsation, then the diagnosis gains still more in probability. It is further supported if, by one of the already described methods (see p. 23), secretion is aspirated and made visible in the previously clean nasal cavity. But we must admit that the appearance of pus on the application of aspiration by the method described does not necessarily point to the inflammation of any particular cavity. Diagnosis can only be assured if, by probing or syringing an accessory cavity, and then, after the probationary cleansing of the nose, we find pus. If a probationary syringing through the natural opening is difficult or impossible, then an exploratory puncture, followed by aspiration or cleansing, will be decisive.

Transillumination is not absolutely reliable (see p. 30), but it is always worth a trial, and will aid the diagnosis.

This will become a puzzle in all those cases where a *combined empyema* exists, *i. e.*, in suppuration of several accessory cavities; and it will present the utmost difficulties of solution if several accessory cavities are diseased on both sides. In such cases we will perhaps succeed in ascertaining from where the pus comes by exclusion of the cavities concerned seriatim. A cavity can be excluded in the following way: A little pad of cotton-wool is pressed into or over the respective ostia. This segregation, however, of the various cavities is rather difficult, and there remains nothing else but to search for the pus in the several cavities by the methods of puncture just described (see above).

It is well, in performing this exploration, to follow a certain plan or order. If pus appears to collect *in the hiatus, laterally or immediately below the middle concha,* it points to an affection of the *sinuses situated towards the front* (antrum of *Highmore,* frontal sinus, anterior ethmoidal cells). In such a case the maxillary cavity, which is that most commonly affected and easiest accessible, must be explored first, and the ethmoidal cells next; and, lastly, the frontal sinus, which offers the most difficulties. If the pus seems to collect in the more *posterior parts of the nasal cavity,* or in the *naso-pharyngeal space or olfactory chink, i. e.,* if it appears somewhat *above and mesially from the middle concha,* our attention must be directed towards the upper posterior cavities (posterior ethmoidal and sphenoidal cells), and we must attend first to the sphenoidal sinus, and then finally to the posterior ethmoidal cells.

Most probably the diagnosis of a combined empyema will not be assured before an operation on one of the numerous cavities has already been commenced.

In cases of foetid discharge a differential diagnosis must be made between foreign bodies, nasal calculi, tumours, and tertiary syphilis. Acute nasal catarrh, though very often accompanied by purulent discharge, probably never shows foetor ex naso.

Prognosis.—Acute inflammatory processes, albeit with copious purulent discharge, for the most part recover spontaneously. The chronic inflammatory affections are cured or, at least, much ameliorated by proper treatment, complications not being often observed. But it has happened that in chronic cases the inflammation has spread into the neighbouring tissues and organs (eye, orbit, etc.), or, by encroaching on the cranial cavity, has led to death from meningitis.

Treatment.—In acute suppuration of the accessory cavities the best treatment is the expectant method; and the physician must proceed symptomatically, just as in a case of acute rhinitis. In chronic suppuration our treatment must be strictly individual, and it is highly objectionable to operate without distinct indication. It is just in the field of suppuration in the accessory cavities, that, encouraged by the progress of diagnosis, a more radical method of treatment has been adopted, which oversteps considerably the limits drawn by functional needs or cosmetic considerations. Life is, as we have already said, very little endangered by complications, and it is just the radical operations which have resulted in intracranial affections. Besides, the gain by radical operations is often finally not greater than by more conservative methods. We should, therefore, attack chronic suppuration, which causes little or no trouble, only by mild douching or intranasal operative methods. To lay bare the suppuration, after the methods described in the following section, is only permissible where the patient complains of serious trouble, and where he is prevented from following his occupation, or complications are threatening. In this sphere surgical technique is still tentative, and a wise and determined restraint is here desirable, because of the many methods of operation. It is certain that much has here to be modified, corrected, or eliminated, as numerous questions still wait to be solved.

(*b*) SPECIAL.

1. INFLAMMATION OF THE ANTRUM OF HIGHMORE.

Etiology.—The causes of inflammation have already been indicated in the general section. (See p. 132.) Here we only desire to show the rôle the teeth play in the etiology of the inflammation of the maxillary cavity. The anatomical relations between the floor of the antrum and the dental alveoli described in the general section (see p. 6) explain clearly why inflammatory processes about the dental roots, *e. g.*, alveolar periostitis, dental cysts, or abscesses, etc., so often lead to infection of the maxillary cavity. Most commonly the fangs of the first and second molar teeth set up the trouble. Unclean instruments used for extraction of the teeth may be the cause of an infection, and intranasal operations likewise. I have myself observed an acute suppuration of the antrum on which I was called upon to operate which followed a partial resection of the middle concha.

Chronic inflammation of the maxillary cavity, in the majority of cases, follows on the acute stage, under conditions already discussed.

Symptoms.—It may be taken for granted that not a few acute or chronic inflammations of slight degree show clinically little or no symptoms; in yet other cases, however, the symptoms or signs are unmistakable. These symptoms are: (1) local; (2) general.

Local symptoms are manifested by a feeling of tension in the affected region, sometimes combined with tenderness in the facial wall. Radiating pain occurs frequently on blowing the nose, coughing, or on stooping; the nose is obstructed and there is epiphora (lachrymation). Discharge, mostly muco-purulent, or, if the inflammation is caused by carious teeth, a discharge of foetid secretion from the nose, may be noticed. The discharge is not continuous, but periodical, and is usually followed by pain and tension in the nose being relieved. At other times it appears if the patient bends the head towards the healthy side. The constant irritation kept up by the suppuration gives rise to swelling of the mucous membrane, polypoid growth, hypertrophies, etc., in the vicinity of the ostium maxillare; in other cases atrophic processes prevail, or one finds hypertrophy and atrophy vying with each other. In very chronic cases, if the maxillary ostium is blocked, the nasal wall of the antrum is liable to bulge.

General symptoms, fever, giddiness, weakness, and so on, are rarely absent if inflammation is pronounced.

Course.—The course is variable. Acute inflammation of the maxillary sinus often recovers spontaneously in two to three weeks, but not infrequently it passes into the chronic stage. In chronic inflammation changes for the better or worse follow each other, and exacerbation often follows acute nasal catarrh. Complications are rare. Sometimes the abscess perforates into the orbit; at others, there is exophthalmos, contraction of the field of vision, or amblyopia occurs.

Diagnosis.—In pronounced cases, and particularly in acute ones, the disease is easily recognized by the apparent symptoms. The diagnosis depends on this and on the result of rhinoscopic examination. The purulent secretion mostly appears in the hiatus semilunaris and flows down over the middle meatus and lower concha to the floor of the nose; or if the bulla ethmoidalis is of a large size or the middle concha enlarged, the discharge may flow backwards into the naso-pharyngeal space. In long-standing suppuration the mucous membrane covering the processus uncinatus appears conspicuously swollen and the lateral fold of mucous membrane (*Kaufmann*), under these circumstances, sometimes pushes the middle concha inwards towards the nasal septum. In some cases the method of aspiration and transillumination will prove useful, and in doubtful cases an exploratory puncture or probationary washing-out will help.

The two latter methods, moreover, serve as useful therapeutical measures, especially in the acute condition.

The washing-out of the maxillary cavity should be done, if possible, through the natural opening, and must be preceded by examination with the probe. (See Fig. 50.)

For this purpose a rigid probe is used, which is bent to a right angle 1 cm. from the end. Having previously anaemized and anaesthetized the whole lateral wall, the middle meatus is cautiously explored with the probe until we reach the infundibulum, into which the end of the probe sinks. If, now, the probe is slightly and very gently shifted forwards or backwards, one can feel how the probe sinks still deeper and is

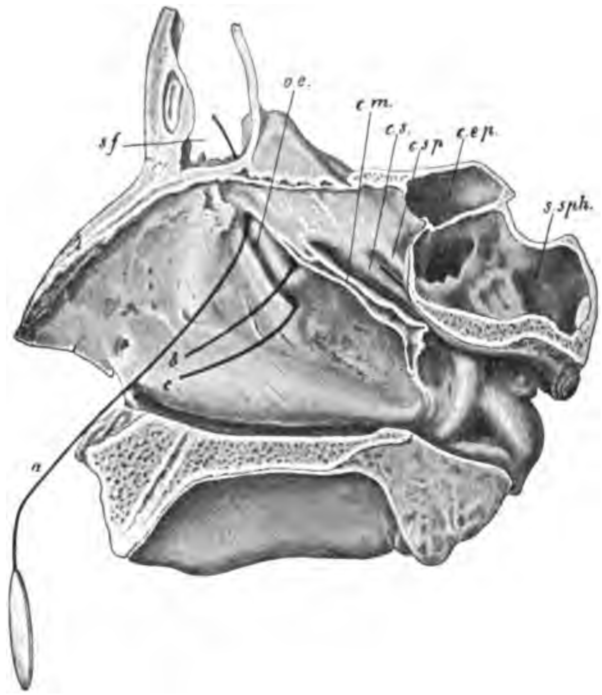


Fig. 50.—Probing of the frontal and maxillary sinus and of the anterior ethmoidal cells (*after Hajek*): *a*, Position of the sound in the frontal sinus; *b*, in the anterior ethmoidal cells; *c*, in the maxillary cavity; *s. f.*, frontal sinus; *o. e.*, ostium ethmoidale; *c. m.*, middle concha (cut); *c. s.*, superior concha; *c. s. p.*, concha suprema; *c. e. p.*, posterior ethmoidal cell (opened); *s. sph.*, sphenoidal sinus.

caught at a particular spot; this is the ostium maxillare. The probing of an accessory foramen is sometimes much easier than that of the infundibular opening. Probing is followed by the introduction of *Hartmann's* tube in the same manner as was followed in the probing (Fig. 51).

The conical end of the catheter is joined to an india-rubber tube, which, after the introduction of the catheter, is connected with a syringe or india-rubber bag. The patient's head is bent slightly forwards, and under low pressure lukewarm sterilized water, to which a little common salt or boric acid or permanganate of potassium may be added, is slowly syringed in.

Exploratory puncture will be resorted to where washing out through the natural or accessory opening is inadmissible, by reason of the narrowness of the hiatus semilunaris; abnormalities in the configuration of the middle meatus; hypertrophic swelling of the middle concha; deformities of the septum, or smallness and unfavourable position of the natural openings. One will sometimes succeed in these cases if the anterior end of the middle concha is resected by means of the snare, scissors, or bone forceps (*Grünwald's*), a proceeding which can be recommended, as it also serves a therapeutic purpose and is often sufficient to secure the permanent discharge of the secretion.

Fig. 51.—Catheter tube for washing out the maxillary cavity (*after Hartmann*).

Exploratory puncture ought always to be made from the *lower meatus.* A small pad of cotton-wool soaked with 20 per cent. cocaine is pressed into the lower meatus and against the lateral wall of the nasal cavity. Having thus made the mucous membrane insensitive and tested the insensibility, a cannula, sufficiently long, thick, and curved, is here introduced as far as the middle of the lower meatus, and tilted upwards as far as possible towards the attachment of the lower concha, and here, about 3 cm. from the anterior end of the lower concha, is thrust through the lateral bony wall, which at this spot is very thin; and now, by slight movements, it is made sure that the point of the cannula is actually in the maxillary cavity. (See Fig. 52.)

Puncture is difficult or impossible if it is tried at a spot too far forwards, because the bony wall is there too thick. Bad accidents, *e. g.*, thrusting the cannula into the opposite wall (the cavity being small) or perforating the orbit, are very rare.

The puncture might be combined with aspiration by attaching the cannula to a *Pravaz* syringe of a capacity of 2 c.c. In a case where the secretion is very thick or folds of the mucous membrane are aspirated, the whole procedure of aspiration fails, and in such a case washing-out has to be performed. After washing out, air, by means of a syringe or india-rubber bag, is pressed into the maxillary cavity for so long a time until the cleansing fluid is emptied out and the cavity dried.

Prognosis.—The prognosis is good in the majority of cases, and chronic inflammations also heal up under proper treatment, or, at least, are so much improved that the patient ceases to complain. In chronic cases, however, it must be admitted that the process of healing is very slow, even after radical operation.

Treatment.—The treatment depends essentially on the duration of the disease.

(*a*) In *acute inflammation of the maxillary sinus* the expectant symptomatic treatment, as already indicated, is mostly sufficient, but if it does not succeed after a certain time, we must, in order to prevent the case becoming chronic, take into consideration certain measures which we have already mentioned as valuable in regard to the diagnosis. These measures are the washing out of the sinus and the partial resection of the middle turbinal.

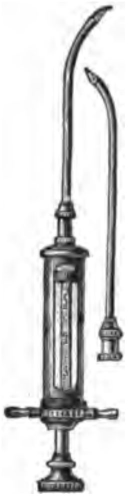


Fig. 52.—Syringe for the exploratory puncture of the maxillary cavity (*after M. Schmidt*).

To remove the anterior end of the middle turbinal the nasal scissors are introduced into the small space between the lateral wall and middle turbinal, and, with the blades directed obliquely backwards, so as not to injure the lamina cribrosa, a piece of the bony attachment of the turbinal (*a–b*, Fig. 53) is cut through.

The guide-tube with the snare bent down is then pushed into the space formed by the cut (*b*), and the loop is thereby adjusted into position (as *b c b*), and then tightened. In great hypertrophy of the anterior end the snare can, however, be passed over the turbinal without any previous cutting. As a last resort, a piece of the uncinated process also must be resected by thrusting pointed scissors through the middle of the process,

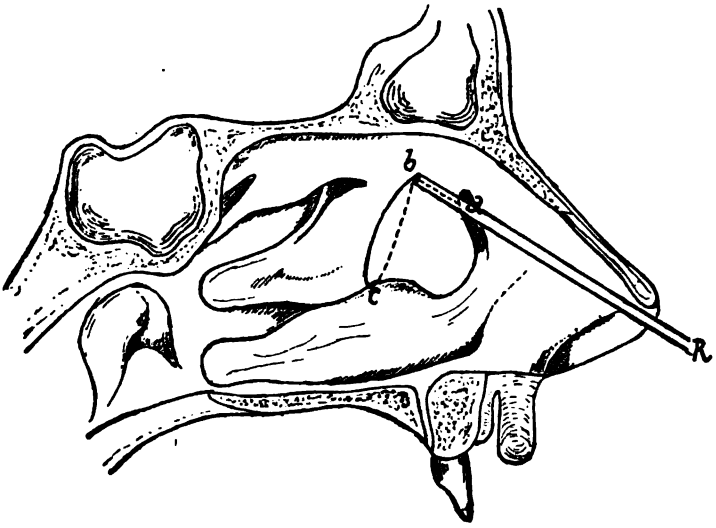


Fig. 53.—Resection of the anterior end of the middle turbinal (diagrammatic): *a–b*, Cut by scissors in end of middle turbinal; *b–c*. . .*b*, position of the snare passed over the turbinal; *r*, guide-tube for the snare.

and cutting upwards and downwards the lateral fold of the mucous membrane with its bony support.

In cases where the secretion is very profuse or thick, the ordinary cannula of 1½ to 2 mm., which is used for washing out, is not suited to drain the antrum. This being the case, the opening in the inferior meatus is made with a larger sized trocar (see Fig. 54), or the sinus can be attacked from the outside through the canine fossa. If the suppuration is caused by a carious tooth, this must be extracted, and the antrum can then be opened through the alveolus. These three methods will be discussed later on.

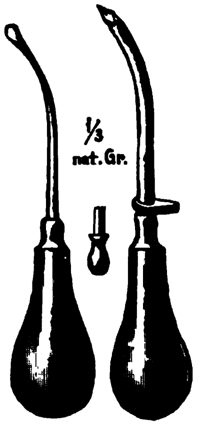


Fig. 54.—Trocar for maxillary cavity.

(*b*) *Sinusitis Maxillaris Chronica* (*Chronic Inflammation of the Maxillary Cavity*).—The treatment of chronic inflammation of the maxillary sinus is, in the first place, directed to delivering the sinus of its contents. This is done, as already indicated, by washing out per vias naturales (p. 140), and ultimate impediments, for instance, hypertrophies, polypoid growths, enlarged portions of the conchae, must be removed. If the washing out through the natural opening prove difficult or impossible, the *antrum must be opened in the inferior meatus* by means of a trocar (*Mikulicz, Krause, Noltenius, Halle*) by the same way as we have described for the exploratory puncture. Some authors advise making the opening in the middle meatus. The inferior meatus, however, has this advantage that the punctured opening can be enlarged, if it be desired. For this latter purpose curved punch forceps are used, which remove the lateral wall by cutting backwards from the opening (*Sonnenthal, Spiess, Wagener*). For this purpose a very practical type of punch forceps is designed by *Onodi*, which has a sharp point at the tip and so can be used also as a trocar.

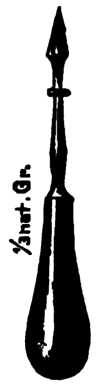


Fig. 55.—Gimlet for dental alveolus.

All the methods of washing out described till now have this drawback, that the patient himself can seldom learn how to perform it. For him it is much better if the cavity is opened from the alveolus of an extracted upper molar tooth by means of a special gimlet (*Hartmann, Hajek*) under local anaesthesia. This method is specially advisable in cases where the second bicuspid or the molar teeth are carious and have to be sacrificed; and, as a last resort, a healthy tooth has to go. The extraction is followed by the opening of the alveolus, and then afterwards the patient is able to treat himself unaided. The small aperture, however, such as that made in the inferior meatus, is not easily kept open. For a similar reason a small india-rubber stopper must be put into the alveolar opening, which keeps it patent and at the same time shuts it off.

In flat palates and wide alveolar processes *Jurasz* adopts the following method: Under local anaesthesia by cocaine and adrenalin, he makes a broad opening into the antrum from a carious upper bicuspid or molar tooth by means of a dental drill, and, if necessary, cutting away a small part of the facial wall of the upper maxillary bone. The opening is occluded with a sterilizable plate of sheet iron, which is only taken off for the purpose of washing out. This "radical" opening of the alveolar process is said to enable the introduction of the little finger for purposes of exploring and for scraping out the antrum with a small scoop.

Under these methods of washing out, which at first are done daily, later every second day, many cases heal completely in a shorter or longer time. Unfortunately, in very numerous cases the mucous membrane is so much altered that only a *broad opening from the outside through the canine fossa can be successful.* Only by this latter method are inspection of the cavity or examination with the finger and radical removal of polypi, granulations, etc., possible. This method, first described by *Desault-Küster*, has latterly been modified.

The operation is done under local or general anaesthesia.

The angle of the mouth is drawn outwards and upwards; a big swab is put at the back, in the pocket of the cheek; the mucous membrane is then cut through horizontally above the alveolar margin down to the bone; the periosteum is then pushed back with a raspatory, care being taken not to injure the infra-orbital nerve, and a hole is made in the thin bony wall with a gouge. Having stopped the haemorrhage, which is frequently copious, the aperture is enlarged with a *Hajek-Claus* punch forceps (Fig. 56), especially outwards and upwards towards the zygomatic process. This done, the cavity is inspected and palpated in turn and all pathological matter removed by the curette. Healthy mucous membrane should not be taken away.

The cavity is now thoroughly cleaned and filled with xeroform gauze, or, in case of marked foetor, with iodoform gauze; but not too firmly, so as not to cause too great an oedema of the cheek. The first tampon remains in from four to five days. The after-treatment consists in regularly washing out, at first daily, later on, every second or third day, and then always following it by packing with conical tampons enveloped in gauze. These tampons allow the cavity to contract, but are sufficient to keep it patent and to permit effective control.

Unfortunately, the *Desault-Küster* method of operation is also not free from blame, which, however, does not concern the operation itself, but the after-treatment, in that it takes a very long time before cicatrization is complete and a cure established. Sometimes a fistula remains, which refuses to close of itself. There is no doubt that this is due to the constant irritation kept up by the repeated packing. The after-treatment, by continued packing of bony cavities with rigid walls, opened by operation, only too often means a hopeless fight against the oft-recurring granulations. We must, therefore, make it a rule not to pack too firmly and for too long a period. It would be the best to operate in such a way that after radical curetting the post-operative packing could be avoided, without hindrance to the outlet of the secretion.

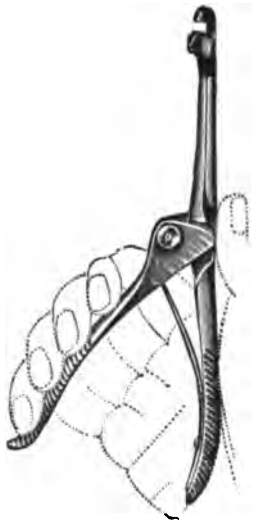


Fig. 56.—Long punch forceps for the maxillary cavity (*after Hajek-Claus*).

These requirements are fulfilled by the method of *Caldwell-Luc*, by which the maxillary cavity, opened from the canine fossa, is shut towards the mouth and drained into the nasal cavity. The facial wall is freely opened from the canine fossa, and the floor curetted to remove all diseased tissue, as previously described; the lower part of the inner wall of the maxillary cavity, which corresponds to the inferior meatus, is now removed by light strokes with a chisel. Care must be taken to preserve the mucous membrane. From within the nose a blunt probe is introduced into the inferior meatus as far in as possible, and the mucous membrane is pushed outwards towards the maxillary cavity; the protruding portion is then incised and the cut carried through the mucous membrane, correspondingly with the attachment of the lower concha. This done, a vertical cut is made in front and behind down to the floor of the maxillary cavity; and so a flap of mucous membrane, formed in the region of the lower meatus, is reflected towards the maxillary cavity, thereby covering its floor. The part of the lower concha in front of the opening is then cut away by scissors, and the maxillary cavity loosely filled with gauze, the ends being brought out through the nose. The oral wound is closed by interrupted sutures, which may be removed after eight days, and the gauze may also remain for eight days or longer. No further packing is necessary, and instead of it, iodoform may be insufflated several times, or, if need be, the cavity can be washed out.

Although all irritative manipulation is avoided by this method, nevertheless, it takes a long time before the cavity is covered with new epithelium. Besides this, there is the disadvantage that the field of operation is not as open to inspection as in the former method. Infection from the nose, as we know by experience, is little to be feared.

2. INFLAMMATION OF THE FRONTAL SINUS (SINUSITIS FRONTALIS).

Inflammatory processes in the frontal sinus almost always involve the anterior ethmoidal cells, and this is not surprising when regard is taken of the intimate relation of the frontal sinus with the ethmoidal cells. (See p. 8.) We should, therefore, as *Killian* points out, speak rather of cellulo-sinusitis frontalis.

Etiology.—The etiology is the same as in acute inflammation of the maxillary sinus, with the exclusion of dental causes. Chronic inflammation usually originates from the acute, if unfavourable local conditions prevent the escape of the secretions from the inflamed frontal sinus.

Symptoms and Course.—The *symptoms* are, *mutatis mutandis*, very similar to those of the maxillary cavity. Characteristically frequent, however, is the agonizing frontal headache, which occurs in the acute or acutely exacerbated chronic cases; sometimes, like attacks of neuralgia, in the first part of the day, and aggravated by every movement of the head, rendering the patient quite incapable of any physical or mental work. In exceptional cases pain is absent. The region of the forehead, and especially the upper and inner angle of the orbital wall, is often abnormally tender, and sometimes the skin over the eyebrow is red and oedematous.

The secretion varies in quantity. If the exit be blocked, it might even entirely cease, an occurrence which usually aggravates the headache.

The eye and orbit are mostly implicated; there occur epiphora, photophobia, asthenopia, etc.

The *course* is mainly determined by the complications, which, though on the whole infrequent, yet occur oftener than in inflammation of the antrum of *Highmore*, and are accounted for by the peculiar anatomical relations of the frontal sinus. Complications are: (*a*) Distention and dilatation of the sinus; (*b*) ulceration and perforation of the wall and spreading into the vicinity.

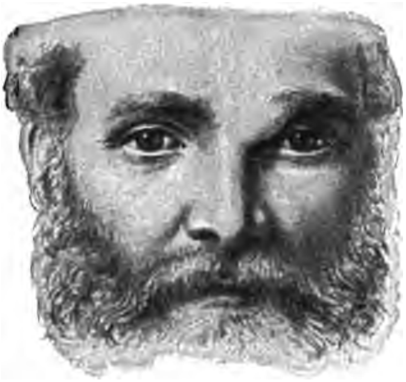


Fig. 57.—Chronic empyema of the left frontal sinus, showing well the protrusion of the upper and inner orbital wall (*after Hajek*).

(*a*) Dilatation of the sinus is observed only in the chronic cases. It is produced by the exit becoming permanently or periodically blocked. The orbital wall is mostly concerned, and bulges forwards like a tumour and displaces the globe downwards and outwards. The protruding part of the bone becomes thinned by absorption, and this sometimes so much that it crackles like parchment. (See Fig. 57.)

The contents of the dilated sinus are rarely serous (hydrops), oftener mucoid, or purulent (mucocele or empyema).

(*b*) Ulceration and perforation have also been observed in the acute stage; and this was sometimes followed by spreading in the neighbourhood as the result of thrombosed veins. The most liable spot here also is the orbital wall. Much less frequently is the anterior wall implicated, and, fortunately, still rarer is the cerebral wall affected. The sequelae are fistulae, phlegmon of the orbital cellular tissue, displacement of the eye, with diplopia, and immobility, oedematous swelling of the lids, exophthalmos, etc. But meningitis also, and abscess of the brain, as well as thrombosis of the cavernous sinus, have been observed.

Diagnosis.—In acute cases frontal headache, especially if localized just above the nose, with tenderness of the upper and inner angle of the orbit, will direct our attention to the frontal sinus, the more so if the patient is suffering or has suffered recently from acute rhinitis. On examination of the nose by rhinoscopy we notice the pus running down from the anterior and upper part of the middle meatus. The diagnosis may be completed by the probe.

In chronic cases the diagnosis is often very difficult. Confusion with trigeminal neuralgia, hysteria, gummatous periostitis, inflammation of the maxillary and ethmoidal sinuses, and malignant tumours have frequently occurred. Transillumination is quite unreliable. In order to make a differential diagnosis between maxillary or frontal sinus disease one should notice if pus escapes more readily in the inclined or in the erect position of the head; in the first case, it is the antrum of *Highmore;* in the latter case it is the frontal sinus which is affected. But this examination is not free from error, and the method may entirely fail if the respective ostia are blocked or if there is a multiple affection. We ensure the diagnosis, however, by probing the frontal sinus. Under local anaesthesia with cocaine and adrenalin a probe bent at its anterior end is pushed laterally from the anterior end of the middle concha straight upwards. If, now, the handle of the probe be lowered, the upper end can be directed more towards the front and the middle line; all these movements should be done gently and without using any force. In order to ascertain if the probe is actually in the frontal sinus (see Fig. 50), the manoeuvre is repeated in exactly the same manner with a second probe, bent in the same way and of identical length and curves, outside, on the dorsum of the nose, and then compared as to direction and length from the nostril upwards, and then notice is taken as to whether the end of the external probe lies above the orbital margin. This manoeuvre can be controlled by x-rays.

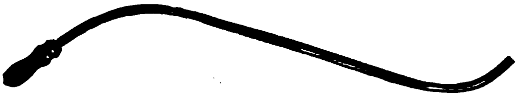


Fig. 58.—Catheter for washing out the frontal sinus (*after Killian*).

Difficulties Met With in Probing.—With respect to the anatomical relations between the frontal sinus and ethmoidal cells, we shall not be surprised to find that the probe is easily caught in one of the cells, an event, however, which does not count for much, especially if pus is found, for the reason that suppuration of the frontal sinus, as has been said, is nearly always accompanied by implication of the infundibular cells. Greater difficulties arise in probing from any marked deviation of the septum, from an excessively large bulla ethmoidalis, or an abnormally narrow infundibulum.

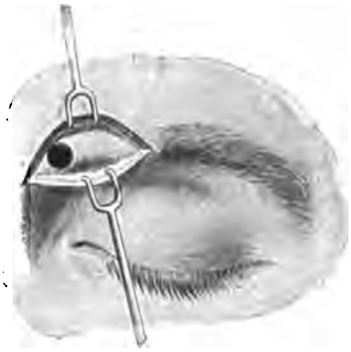


Fig. 59.—Exploratory opening of the frontal sinus in the eyebrow.

The matter will be made easier in many cases by resection of the anterior end of the middle concha, and the removal of excrescences and hypertrophies.

The diagnosis is certain if pus runs along the probe, or if, on syringing, pus is found in the return flow. In rare cases only the sinus has to be opened from without.

This is done in the following way: A cut is made in the eyebrow itself (on the margin of the tuft of hair), beginning at the incisura nasofrontalis, and is carried along for from 3 to 4 cm., cutting through all the soft parts down to the bone; the periosteum is now pushed upwards towards the glabella with an elevator, and a small hole is made immediately below the nasal end of the eyebrow by means of a chisel, carefully advancing as it is deepened. This opening will serve a three-fold purpose: firstly, to ensure a diagnosis; secondly, it is therapeutically of value because it establishes a ready escape to the accumulated pus; and, lastly, in chronic cases it can be easily enlarged for the purpose of a radical operation.

Prognosis.—The majority of cases of acute inflammation heal spontaneously in the course of a few days or weeks. Transition into a chronic condition is relatively rare. It has already been pointed out that complications are here more frequent than in disease of the maxillary cavity. Long-standing and obdurate chronic suppuration can only be cured by means of a radical operation.

Treatment.—(*a*) In *acute inflammation*, as in acute rhinitis, symptomatic treatment is sufficient (see p. 72). In cases of persistent secretion a dessertspoonful of a solution of iodide of potassium 5 : 200 given internally twice or thrice daily will be found useful; and in severe headache hot fomentations will prove very beneficial. In time, aspiration or the resection of the anterior end of the middle concha will start the pus flowing. In very serious cases the sinus must be opened from in front, as described above (p. 149).

(*b*) *Chronic Sinusitis.*—In milder cases we can secure the permanent exit of the secretion by resecting the swollen parts of the mucous membrane in the anterior portion of the middle meatus, especially by cutting away the operculum of the middle concha or an excessive bulla ethmoidalis or existing polypi. Sometimes the opening of the anterior ethmoidal cells will be required; and this is done with a suitably bent pair of *Grünwald's* revolving nasal forceps (see Fig. 60).

By this method pain is instantly relieved and secretion diminished; eventually, resection of the operculum has to follow, and a small catheter is then introduced into the sinus, in order to wash out the frontal cavity regularly; in the same way as is done in maxillary suppuration.

If by this treatment the disease is improved, we have no excuse to abandon it. But if the patient, on account of the prolonged and troublesome manipulations, becomes somewhat impatient and nervous, and if he demands to be speedily liberated from his troubles, or if this intranasal treatment fails on account of the discharge remaining foetid, or on account of complications and acute exacerbations, we must resort to operative treatment, viz., to open the frontal sinus sufficiently freely for the purpose of inducing atrophy (occlusion) after the removal of the diseased mucous membrane. There exist many methods of operation which aim at the resection of either the anterior or lower portion of the frontal wall, or of both of them, in order to afterwards drain, either through the nose or directly outwards. By other methods a flap of bone and periosteum is formed which, after the curetting of the sinus and ethmoidal cells, must be replanted.

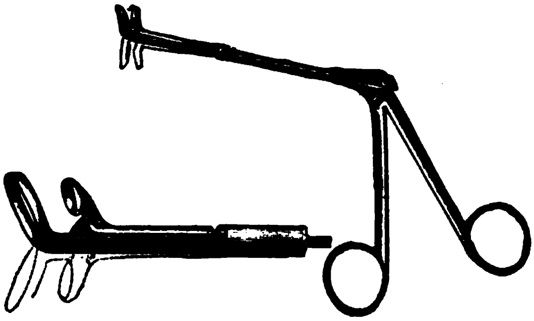


Fig. 60.—*Grünwald's* rotatory nasal forceps.

By Killian's radical operation the frontal cavity is made into one single flat groove by cutting away all the septa and partitions, and overhanging or obstructing parts. This demand, indispensable as it is from the surgical point of view, is fulfilled by sacrificing the anterior and lower portion of the frontal sinus and by the resection of the frontal process of the upper maxillary bone. In order to avoid a later sinking-in of the forehead and the consequent disfigurement, the upper margin of the orbit is left as a small bridge of bone.*

In deep radical sinus operations, the cosmetic effect leaves much to be desired, but we possess in the injection of paraffin a means of improving a later consequent depression of the forehead. Some authors, however, deny the value of these injections because the paraffin in time becomes absorbed or altered. In order to avoid any depression whatever of the forehead, *Ritter* has proposed to always open the frontal sinus from the orbit, and to make only a small opening in the outer facial wall, at a point corresponding to the highest point of the cavity. The sinus is then scraped out with a suitably curved scoop.

Various writers have pointed out that atrophy (occlusion) of the frontal cavity is not necessary for the establishment of a permanent cure, and that less radical operations can claim the same good result. Indeed, in not a few cases operation, after *Luc-Ogston,* may prove sufficient, and the forehead keep its normal configuration, as follows: A cut is made along the inner third of the orbital margin; the periosteum is pushed towards the forehead and orbit; the frontal sinus for about 1 cm. (or a little more) in diameter, is opened a little laterally from the middle line; the opening is then followed by a thorough enlargement of the nasofrontal duct, and the opening of all the anterior ethmoidal cells which may lay in front of it, by means of the sharp scoop. Having thus established a free communication between the frontal and nasal cavities, a drainage-tube or tampon is put into the nose from the frontal sinus, and the external wound closed. The dressing may be left in situ for eight days.

3. INFLAMMATION OF THE ETHMOIDAL CELLS (CELLULITIS ETHMOIDALIS; ETHMOIDITIS).

Etiology.—As to the cause, we again refer the reader to what we have already said in the general section of this chapter.

Pathology.—The pathological changes also reflect generally the conditions related in the general section.

It should be noted that, owing to the greater destructibility of the mucous membrane, the bony parts of the frontal cavity are liable to become involved early in the attack, so that hyperplastic and atrophic processes, and, more rarely, necrosis, are relatively soon encountered. It is also remarkable how easily the mucous membrane, owing to its loose structure, can become swollen by oedematous infiltration, which is the most common cause of the growth of polypi. Hence the almost regular occurrence of polypi in the longer standing cases of inflammation of the mucous membrane of the ethmoidal sinuses. Inflammation and oedematous infiltration of the ethmoidal mucous membrane, however, are not always due to an irritation caused by secretion of an accessory cavity flowing over it, as there are many other causes. The presence of nasal mucous polypi, therefore, often, but not always, indicates a suppuration in the accessory cavities.

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* For further details see Archiv für Laryngologie, 1902, vol. xii, and Verhandlungen süddeutscher Laryngologen, 1904.

There are certainly cases where at no time an accessory suppuration could be discovered; this means that the formation of polypi is the outcome of a general inflammation of the nasal mucous membrane (rhinitis hypertrophica) (see p. 75). In the latter case the polypi are not quite so numerous, but, as if to make up for the smaller number, are of larger size and show little inclination to recur after removal. On the other hand, in suppuration of the accessory cavities the appearance of a great mass of small or the smallest polypi, with a great disposition to recur, is conspicuous. Between these numerous polypi pus is steadily oozing, whereas in non-suppurative or simple polypi pus is scanty or entirely absent.

One single cell alone may be affected at one time, or the hypertrophied middle concha itself, or the bulla ethmoidalis; less often the anterior and seldom, seemingly, the posterior cells may become affected. In some unique cases the entire ethmoidal labyrinth might be concerned.

Symptoms.—In inflammation of the ethmoidal cells, especially in the chronic form, the subjective symptoms are little marked. On examination one will sometimes find tenderness over the root of the nose, and perhaps, in long-standing disease, some distention.

In suppuration of the posterior cells, but also in that of the anterior, if the pus has to make its escape backwards because of the blocking of the anterior passage, the signs of the naso-pharyngeal catarrh, kept up by constant irritation by purulent secretions, are prevalent; and that is why we should be always on the lookout for suppuration of an accessory cavity in every case of naso-pharyngeal catarrh.

Diagnosis.—The diagnosis depends, of course, on the rhinoscopic results, and, as regards this latter, it is often erroneous, especially if there be latent suppuration or if the pus is shut in, *e. g.*, because it cannot escape and perhaps produces distention of the bony walls only (mucocele, empyema). On the other hand, it is just in cases of retained suppuration that we are able to find signs, which unmistakably point in a certain direction; *e. g.*, dislocation of the eyeball, swelling at the inner orbital angle, etc.

In open suppuration, where pus is discharged into the nasal cavity, we must notice, from which part or section of the nasal cavity the pus is derived. If pus shows itself in the middle meatus, when suppuration of the maxillary or frontal sinus can be excluded, then disease of the anterior ethmoidal cells can be assumed, though we must not be afraid to mention that suppuration of the frontal sinus is often combined with anterior ethmoidal suppuration.

We have now to determine exactly the focus of origin of the pus. This can best be done by probing. If the middle concha is found to be swollen, brittle, tender, or even rough; and if the probe, on pushing gently, readily sinks in at this spot, causing pus to emerge; and if, in addition, polypi are met with, the diagnosis cannot be doubtful.

It should be remembered that the uneven-edged, thin, and brittle walls of the ethmoid bone impart also, under normal circumstances, especially on somewhat forced probing, the sensation as of crude bone. A markedly distended bulla ethmoidalis also, in combination with an atrophic rhinitis in the inferior meatus, will direct our attention to a possible chronic suppuration of the ethmoidal cells. It is sometimes only necessary to lift the middle concha somewhat in order to at once see the pus flowing out. In some cases polypi or part of the middle concha must be removed in order to clear the field; and this will also prove of value, as thereby the more are the abscesses opened. It often happens that, on removing a polypus with the snare, the osseous matrix is also torn away, and when the imprisoned pus commences to flow, then we know that we have opened a cave of suppurative disease. Scabs on the middle concha should be removed with forceps, and if pus oozes out, we will probably find its site of origin with the probe.

Sounding the normal typical opening of the anterior ethmoidal cells, in the hiatus semilunaris, only succeeds in rare cases (*Hajek*). If the secretion shows itself in the rima olfactoria, that is, mesially from the middle concha, or in the hinder part of the nose or in the postnasal space, then we have to deal with an affection of the *posterior ethmoidal cells* or of *the sphenoidal cavity* or of *both of them.*

By removing a part of or the entire middle concha we are enabled to follow up the pus to its origin. We should not forget, however, that pus found in the postnasal space may, under certain conditions (see p. 135), come from the anterior cavities.

In some cases the method of aspiration will show us whether the pus is derived from the anterior or posterior section of the ethmoidal labyrinth.

Prognosis.—This depends on the complications or not of other organs.

Bursting into the orbit or even into the cranial cavity may take place in the acute and chronic stage.

Treatment.—The intranasal opening of the cells is the rule; and only in exceptional cases, where complications of other organs require it (fistula or abscess of orbit or brain, etc.), or if the intranasal treatment is inadmissible, is the labyrinth to be opened from the outside.

In combined disease (frontal and ethmoidal suppuration) *Killian's* method of radical operation should be adopted. Otherwise the labyrinth can be opened from the orbit after the method of *Kuhnt* or *Grünwald.*

Grünwald makes an incision immediately below the eyebrow, and, beginning at the middle of it, carries the cut circularly towards the root of the nose; thence he turns downwards to the middle of the nasal bone. The supra-orbital nerve should be saved or pushed aside. The whole flap, containing skin and periosteum, is now pushed downwards with a raspatory, thus laying free the inner orbital wall. Hence, from below, and as much as possible away from the region of the lachrymal bone, the labyrinth is penetrated by means of the chisel. *Kuhnt* operates in a similar way.

The intranasal opening of suppurating cells is frequently the same as we have advised for the purpose of securing a diagnosis (removing of polypi, resection of middle concha and of the bulla ethmoidalis). In localized suppuration, these measures are quite sufficient to establish a permanent cure.

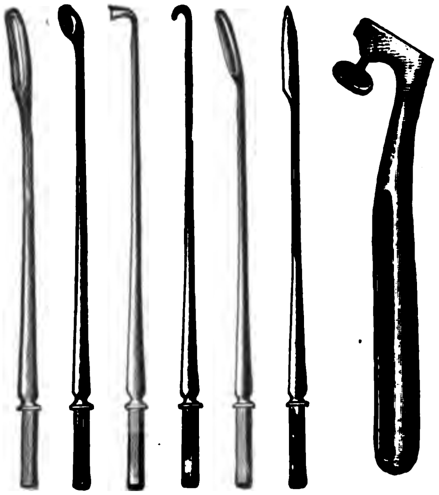

Fig. 61.—*Hajek's* instruments for opening and scraping out the ethmoidal labyrinth.

In other cases the walls are broken by means of *Hajek's* hooks (Fig. 61), the openings enlarged with bone forceps, and the cavity scraped out with a sharp scoop.

These manipulations must, of course, be very carefully done, because of the thinness of the os planum (lamina papyracea). The posterior cells can only be reached after removing the middle concha. Haemorrhage is not considerable, and plugging is nearly always superfluous.

Unfortunately, the intranasal manipulations have to be often repeated, and treatment, which is even continued for months, affords only amelioration, but no permanent cure, because we do not succeed by them in removing the entire mucous membrane lining the ethmoidal cells.

Whether in such cases an opening from the outside will finally be required, depends much on the anatomical conditions, and chiefly on the patient himself (seriousness of symptoms, deformities of the nose, consequent disfigurement, etc.).

4. INFLAMMATION OF THE SPHENOIDAL SINUS (SINUSITIS SPHENOIDALIS).

Etiology and Pathology.—Is mainly of the same nature as in inflammation of the posterior ethmoidal cells, which are very often in direct communication with the sphenoidal cavity. .

Symptoms.—The symptoms equally give little cause for special consideration: the subjective ones, because they are little characteristic save for a dull pain in the back or top of the head; the objective ones, because they resemble altogether those discussed in inflammation of the posterior ethmoidal cells. As in the latter, the usual scanty secretion flows down into the postnasal cavity, there to dry up and form scabs (retronasal catarrh).

The special clinical rank which the sphenoidal sinus holds is in a great measure due to its relation to important structures in its neighbourhood, viz., base of the brain, optic and oculomotor nerves, cavernous sinus, internal carotid artery, etc. Destruction of the walls of a large sphenoidal cavity is apt to cause serious complications, such as meningitis, thrombosis of the cavernous sinus, cerebral abscess, amblyopia or amaurosis, optic neuritis, paralysis of ocular muscles, etc., or even cerebral apoplexy, and sometimes, indeed, the pus may even burst into the ethmoidal labyrinth or maxillary sinus.

Diagnosis.—The diagnosis must depend only on the rhinoscopic results, for the subjective symptoms are far too uncertain. The discharge from the sphenoidal sinus appears correspondingly to the position of its ostium, respectively, in the *olfactory chink*, having run over the middle concha, or in the *nasopharyngeal space*.

With anterior rhinoscopy we succeed only when the nasal cavity is wide, as the olfactory chink, being large, it is possible to see the anterior wall of the sphenoidal sinus with its ostium, with the pus oozing from it. Eventually an apparatus for nasal aspiration may be used, or a probe or small catheter introduced after having previously cleansed the nose. If fresh pus comes down soon after the chink has been cleared of pus, a suppuration of the sphenoidal sinus is probable. We then have still to prove whether the pus is derived from the sphenoidal or posterior ethmoidal sinus. Here the long-bladed speculum of *Killian* (middle rhinoscopy) renders good service, for by its introduction we are enabled to open the rima olfactoria so wide that the sphenoidal ostium becomes visible. Otherwise it would be necessary to resect the middle concha in order to follow up the pus to its origin. It is a good thing to temporarily plug the sphenoidal ostium, or if this cannot be found, the recessus spheno-ethmoidalis. If, afterwards, pus collects in front and behind the plug, a combined suppuration must be present, *i. e.*, of the sphenoidal and posterior ethmoidal sinus.

If we do not succeed by any of these methods in observing directly the outflow of the pus, the sphenoidal sinus must be sounded or washed out, which must also be done if the ostium is visible. (See Fig. 62.)

The path from the anterior opening of the nose to the sphenoidal sinus is marked by a line which passes from the anterior nasal spine backwards over *the middle of the lower margin of the middle turbinal.* By gently pushing the probe in this direction between the septum and the middle turbinal backwards and upwards, the anterior wall of the sinus is reached. The blunt point of the probe must be curved downwards and outwards, for it will the more easily succeed in finding the ostium by this little contrivance, and also avoids the thin lamina cribrosa. In any case, great care must be taken not to push the probe upwards *too much in front of the free margin of the turbinal,* because there will be the danger here of penetrating the lamina cribrosa and entering the cranial cavity. If the probe has advanced in the indicated direction for 7½ to 8½ cm., it might be assumed that the point lies in the sphenoidal sinus.

By anterior rhinoscopy, the secondary changes of the nasal mucous membrane can be noticed. *Polypi are very rare* in sphenoidal suppuration. But frequently the nasal mucous membrane of the mesial face of the middle turbinal and opposing septum is swollen, and in advanced stages shows atrophy and scabs.

Posterior rhinoscopy always shows scabs on the roof of the naso-pharynx, and farther down, on the posterior wall of the pharynx. It is sometimes possible to see in the mirror the outflow of pus from the sinus. Scabs are also met with in the nose, as has been already mentioned, and the foetid smell from these scabs, in conjunction with the advancing atrophy of the mucous

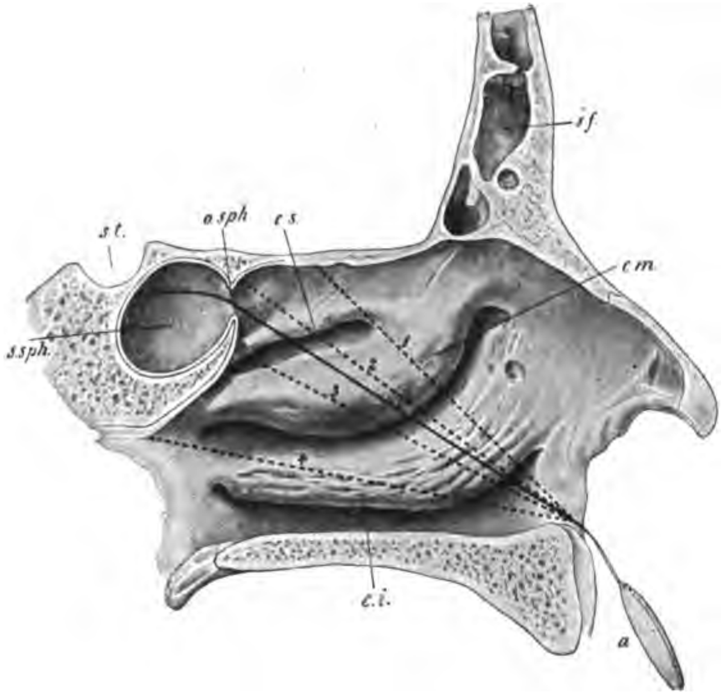

Fig. 62.—Probatory examination of the sphenoidal sinus: *c. i.*, Concha inferior; *c. m.*, concha media; *c. s.*, concha superior; *o. s. ph.*, sphenoidal ostium; *s. t.*, sella turcica; *s. f.*, frontal sinus; *s. sph.*, sphenoidal sinus. The black line shows the right position of the probe, *a;* the dotted lines 1, 2, 3, 4, the wrong directions (after *Hajek*).

membrane, accounts for the complex symptoms, respectively, of rhinitis and of a rhino-pharyngitis atrophica foetida.

Prognosis.—The prognosis is not unfavourable, but is much influenced by the difficulties of diagnosis and treatment met with and by the frequent serious complications.

Treatment.—(*a*) In acute inflammation of the sphenoidal sinus treatment is like that in acute inflammation of any other cavity—expectant and symptomatic. In some cases the method of aspiration should be tried. In other cases, should the complaints continue with unabated insistence on the part of the patient, or should complications become menacing, then the sinus must be opened from in front.

(*b*) In chronic sinusitis the best thing to do is to make a path for the free escape of the secretion by the resection of the middle concha, unless this has already been done for diagnostic purposes, or by the removal of any polypi, or by the method of aspiration. The foregoing procedure will relieve the patient *so much that any further measures are often unnecessary.* Whenever it is possible, washing out should be performed through the natural opening. In some cases it will become necessary to enlarge the sphenoidal ostium, which may be done by pulling with a *Hajek's* hook at the lower edge of the opening downwards and forwards. The hook must be introduced with the point turned backwards and downwards, so as not to endanger the lamina papyracea. Having rendered the cavity more accessible, it should be lightly packed for a week or so, in order to prevent reclosure of the opening, and the edges of the ostium should be cauterized afterwards with trichloracetic acid. Keeping the opening patent is often more difficult than the making of an opening into the sinus in the first place. The secretion often diminishes by this procedure, and assumes a more mucoid character, but hardly ever ceases entirely.

In more severe cases one has to determine on more radical treatment, viz., resection of the intermediary wall of the posterior ethmo-sphenoidal cells (*Hajek*).

Hajek's hook, in the manner described above, is introduced into the olfactory chink, and is pushed upwards towards the upper part of the anterior wall of the sphenoidal sinus. Then, by turning the hook outwards, the inner wall of the ethmoidal sinus is caught, and the lower part of the inner wall, together with the middle concha attached to it, is torn away by a good pull. The broken parts are removed with suitable forceps, and the haemorrhage stopped with swabs. Thus the anterior wall of the sphenoidal sinus is laid open in its whole length. Now, the sphenoidal ostium can be enlarged by the hook, and the whole anterior wall can be removed by way of the ostium by means of *Noebel's* punch forceps (recently modified by *Hajek*), cutting upwards and downwards (Fig. 63).

The cavity is packed daily, and the granulating edges of the opening *slightly* cauterized with lapis infernalis or trichloracetic acid.

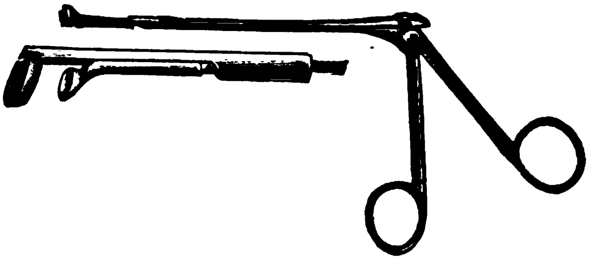

Fig. 63.—Revolving punch forceps for resecting anterior wall of sphenoidal sinus.

The cauterization can be continued every six or eight days until the margins appear thoroughly cicatrized. At the time of cauterization the sinus may be inspected, and all suspicious matter scooped out—care must be taken not to endanger the upper and lateral walls.

By keeping *the sinus open* in this way, the affected mucous membrane may be healed up, or at least put into good condition.

If the sinusitis is combined with empyema of the frontal sinus, the radical operation (after *Killian*) (see p. 151) can be performed.

II. NEW-GROWTHS.

BENIGN TUMOURS.

Besides the polypi, which, strictly speaking, are not new-growths, but the products of inflammation, the tumours which we now have under our consideration are cysts. They often occur in great numbers, and frequently in association with inflammation of the cavities; but appear also independently of inflammation; at least, there are many cases on record where inflammation could never be discovered, or was already abated, long before the appearance of the cyst. Sometimes the maxillary cavity is entirely occupied by one single large cyst.

They form translucent, yellowish or whitish, sometimes pedunculated tumours, of spherical or hemispherical shape. They contain a serous or more or less viscous fluid, many cellular elements, and detritus.

The condition, known as hydrops of the antrum of *Highmore*, viz., the free accumulation of serous fluid in the maxillary sinus, is probably always due to cystic formation, if not identical with it.

In the frontal sinus hydrops and mucocoele have been repeatedly observed, more rarely, cysts. It has happened that cystically degenerated anterior ethmoidal cells grew into the frontal sinus. The *maxillary* or *dental cyst* must not be confounded with the true cysts, of the mucous membrane. They are mostly congenital cysts, which, if unchecked in their growth, push the bony wall forwards, either towards the mouth or towards the face. In the latter case the facial wall might become so thin that it shows signs of parchment crackling, or the cyst bursts through into the canine fossa.

Symptoms.—The symptoms are little marked. If a *large cyst has burst, a large quantity of serous fluid might* be discharged from the nose.

Diagnosis.—Diagnosis offers some difficulties in making a distinction between empyema, mucous cysts of the membrane, and alveolar (maxillary) cysts. The formation of a fistula in the canine fossa points rather to maxillary cyst than to empyema of the sinus; so also does bulging of the facial wall. In the latter case one should not overlook a possible malignant tumour. We must make sure that the sinus is sufficiently opened.

Hydrops and mucocoele of the frontal sinus frequently expand towards the orbit, displacing and dislocating the globe, as does also empyema of the frontal sinus.

Treatment.—If the cysts give rise to serious symptoms, they must be removed by a free opening from the outside.

To the benign tumours belong also the osteomata, which originate preferably in the frontal sinus, but occur also in other cavities.

They grow very slowly, and if of larger size, displace the neighbouring organs (eye, etc.). Usually they exist without symptoms, and are not noticed until they have already attained considerable size. They are then easily recognized by their hardness and lumpy surface. Treatment can only consist in their removal, which is not always an easy matter. Occasionally fibromata, papillomata, and angeiomata are found in the accessory cavities.

MALIGNANT TUMOURS.

The maxillary and frontal sinus are the seat of predilection. It is doubtful whether the sinus is always the primary seat of the tumor, for it often comes so late to the notice of the physician that the question cannot be decided. I myself remember a case of sarcoma of the maxillary sinus the origin of which could be traced into the basis cranii. In some cases not the antrum, but the body of the upper maxillary bone, is the origin of the new-growth (mostly periosteal sarcoma).

At the beginning, little will be noticed, and the patients often complain only of neuralgia in the face or teeth or forehead. After a time, when the tumour has grown and thereby destroyed the respective nerves, anaesthesia will be conspicuous, which, taken in connection with the previous neuralgic pain, serves to direct our suspicion to a neoplasm (*Killian*). If, now, an exploratory puncture is negative, and, on the other hand, transillumination gives a positive result, doubt will become certainty. Later on, the new-growth breaks down and causes a terribly foetid suppuration, and the formation of polypi in the nose, behind which can be felt the freely bleeding tumour.

In other cases the facial wall is bulged forwards or burst through, or the tumour grows through the hard palate, or towards the orbit, displacing the eye or the nose towards the healthy side ("frog face").

In some cases the nature of the growth can only be ascertained by microscopic examination of particles excised for this purpose or discharged spontaneously from the nose.

Prognosis.—The prognosis is almost always bad.

Treatment.—If it can be of any avail, early removal is the only possible resource.

III. INJURIES.

The maxillary and frontal sinuses, which are more exposed, are accordingly oftener injured than the ethmoidal and sphenoidal cavities. If the bony walls have met with an accident, haemorrhage often occurs into the cavity and is a fertile soil for all sorts of pathogenic bacteria, resulting in abscess, suppuration, fistula, and necrosis. Sometimes an emphysema, under the skin of the forehead, eyelid, or cheek, occurs after blowing the nose, if the bones have been fractured by accident.

Treatment.—The treatment follows the general rules of surgery.

IV. FOREIGN BODIES AND PARASITES.

Foreign bodies reach an accessory cavity by accident or by the fault of the surgeon. All sorts of foreign bodies have been found, such as bullets, broken knife-blades, broken teeth, splinters, drainage-tubes, tampons, gauze, tents, broken instruments, etc. They mostly cause suppurative inflammation, or do not allow an existing inflammation to recover.

Diagnosis.—The diagnosis must rely on the history of the case, examination by probe, and eventually by x-rays.

Treatment.—If we have ascertained the presence of a foreign body, it is our duty to remove it by the natural opening, either by means of forceps or hook or syringing, etc., otherwise the cavity must be opened by operation.

Parasites.—Parasites, viz., worms and insects, have been found in the frontal sinus, but only in corpses.

V. TUBERCULOSIS AND SYPHILIS.

Tuberculosis.—Tuberculosis of an accessory cavity occurs still more rarely than in the main cavity, and probably is never primary, but mostly an affection which has spread from a tubercular process in the vicinity. Tubercle bacilli have sometimes been found in the pus of an empyema of the antrum.

Syphilis.—Syphilis of an accessory cavity is likewise, in the majority of cases, to be traced to a syphilitic process in the neighbourhood, as, for instance, syphilitic caries of the vomer may easily spread to the anterior wall of the sphenoidal cavity; but it cannot be denied that, in the accessory cavities, syphilis might also primarily appear especially as a gummatous process. This, however, has not yet been recorded. We certainly admit that every suppuration of the antrum or frontal sinus in a syphilitic person must not necessarily be considered as caused by the syphilis.

PART II.

Diseases of the Mouth.

PART II.

Diseases of the Mouth.

GENERAL SECTION.

I. ANATOMY.

The oral cavity is divided into two portions, the smaller anterior one, lying between the lips, mucous membrane of the cheek, and teeth, is called the vestibulum oris; and the larger portion is enclosed, above, by the hard palate, behind by the soft palate, below by the floor of the mouth and the tongue respectively, and is called the oral cavity proper (cavum oris). Both sections communicate by a gap, which lies between the last molar teeth and the coronary process of the lower jaw.

The **lips** are attached to the gums in the median line by a fold of the mucous membrane (frenulum labii superioris et inferioris).

The **tongue** is fixed chiefly to the inner surface of the chin and to the body (corpus) of the hyoid bone, and is in conjunction with the lower jaw in all directions by means of its coat of mucous membrane. One distinguishes the point or tip; the dorsum, divided by the median groove (sulcus medianus), into two equal parts; the lateral borders, and the root, which broadens at the back towards the epiglottis. The tongue is connected with the floor of the mouth by a fold of the mucous membrane, the frenulum linguae. Another fold of mucous membrane connects the middle of the root with the epiglottis, and is called the ligamentum glosso-epiglotticum medium. At both sides of this fold are the valleculae, which again are bordered laterally by the lateral glosso-epiglottic ligaments (ligamenta glosso-epiglottica lateralia).

The entire back of the tongue as far as the isthmus faucium

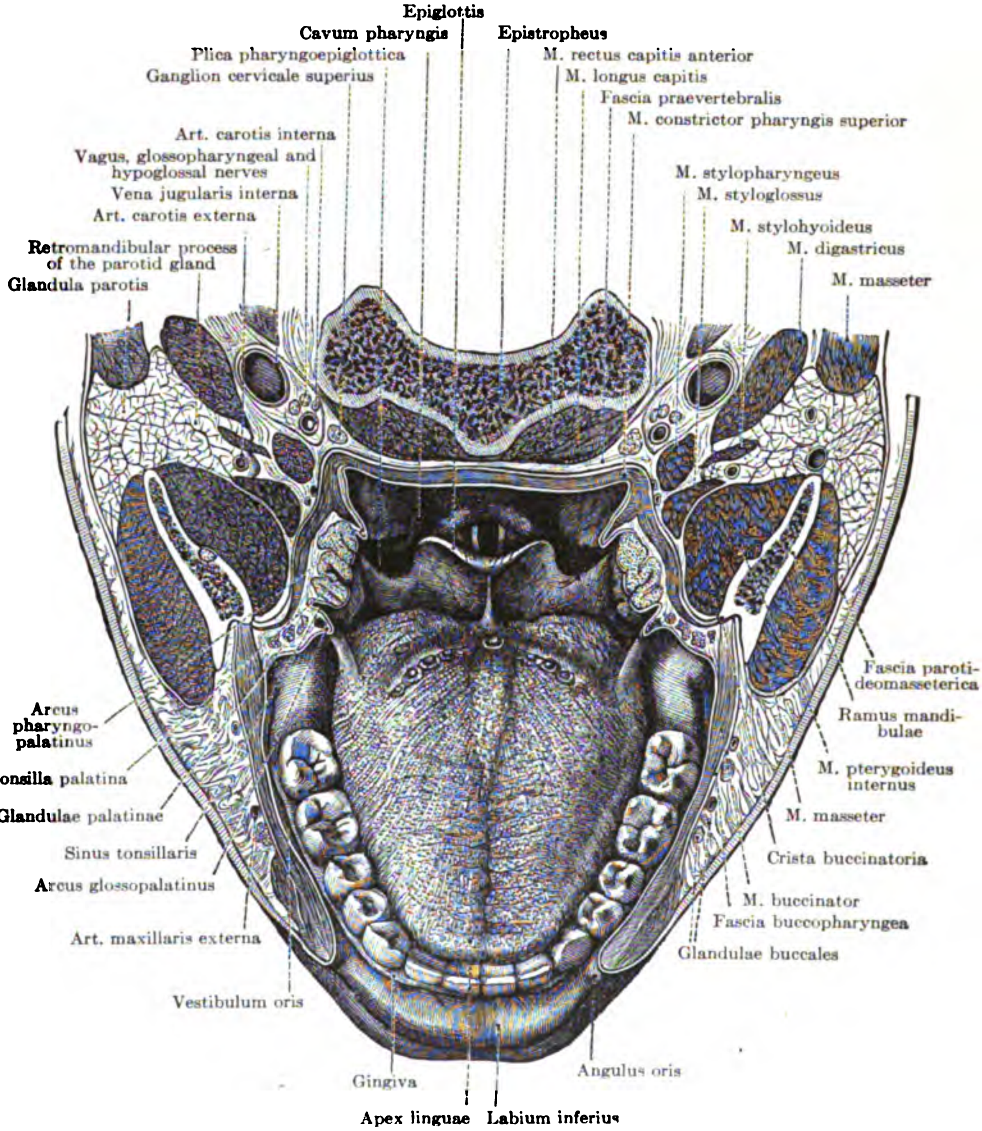

Fig. 64.—Cavum oris (*after Toldt*): The floor of the mouth with the tongue and lower lip, and also the middle section of the head of the pharynx, commonly called "the throat"; the fauces are viewed from above. The cheeks, the tonsils, with the palatine arches, and the lateral and posterior wall of the pharynx, are cut horizontally; the ascending ramus of the lower jaw with the muscles attached to it and the parotid gland are also cut horizontally.

is occupied by the numerous organs of taste and touch, *e. g.*, the small filiform papillae (papillae filiformes), interspersed with the more fungiform or conical papillae (papillae fungiformes), which lend to the surface its granular, villous appearance. The foregoing are limited behind by the large circumvallated papillae (papillae circumvallatae), which are from 8 to 15 in number, and are arranged in the shape of a V, with the apex backwards. Just behind the point of this V the blind foramen (foramen caecum) is found. Further back, towards the epiglottis, lies an accumulation of follicular glands which form a part of the "lymphatic ring" encircling the isthmus faucium. From their acinous structure the glands are sometimes also called the "lingual tonsils" (tonsilla lingualis).

On the lateral margins of the tongue, just in front of the anterior palatine arch, an organ, mostly elevated, flat, or grooved, of the size of a lentil, is found, and which is called the foliated papillae (papilla foliata); it is an organ of taste.

The Muscles of the Mouth.—(*a*) *Muscles of the Lips.*—The muscles which form the lips are either entirely a part of, or enter, or are only inserted into, them. The orbicularis oris muscle is the sphincter of the mouth. Other muscles dilate or open the oral fissure in different ways, thus contributing largely to the mimetic action of the face. The most important of these is the buccinator, also called trumpeter muscle (buccinatorius), for its action of pressing air out of the mouth. The masseter, temporal, external and internal pterygoid muscles serve the action of chewing.

(*b*) *Muscles of the Floor of the Mouth and of the Tongue.*—The floor of the mouth is chiefly formed by the mylohyoideus muscle, which expands like a fan between the two halves of the lower maxillary bone above, being attached below to the hyoid bone; the genio-hyoideus muscle, which lies in the middle line between the chin and the hyoid bone; and the anterior head of the digastric muscle (m. biventer, seu digastricus).

The muscles of the tongue are divided into two groups: The one group, the intrinsic lingual muscle, properly so called, runs within the body of the tongue in longitudinal and transverse bundles (m. longitudinales et transversales), and their action is to modify the shape of the tongue. The other group, genio-hyoglossus, hyoglossus, styloglossus muscles, move the tongue as a whole. Both groups, by variously intercrossing and by joint action, produce the admirable mobility of the tongue which may in some cases be of an acrobatic character, so to speak.

The mucous membrane is lined with several layers of stratified epithelium, and is continuous with the skin on the outer side of the lips; the border is defined by a sharp line, and this lip-margin is called the "red of the lip," because of its colour. On the hard palate and the dental alveoli, where it forms the gum (gingiva), the mucous membrane is firmly attached to the periosteum and cannot be folded. On the middle line of the hard palate it forms a whitish ridge, called the raphé; and on other places the mucous membrane is only loosely attached to the underlying structures. The mucous membrane of the tongue is thin, and is closely attached to it. At the side of the median sulcus or median raphé, on one or both sides, small invaginations of mucous membrane, like culs-de-sac, are found, which, by anxious patients, are sometimes looked upon as defects.

The Glands.—There are very numerous mucous glands in the mouth, and the salivary glands, three in number,—*the parotid, submaxillary,* and *sublingual* glands,—open into the cavity.

The submaxillary and sublingual glands are considered by some authors to be "mixed glands," *e. g.*, glands which produce mucus and saliva.

Blandin-Nuhn's glands are found at and beneath the tip of the tongue, and open onto the fimbriated fold (plica fimbriata), and are still under discussion in respect to their secretion.

The mouths of the *mucous glands* can be seen on the surface as small points of the size of a pin-head, and on the lips their acini are visible through the mucous membrane.

The parotid gland lies in front and below the ear, reaching down as far as the angle of the jaw. Its duct (ductus *Stenonianus*—Stenonian duct) runs forwards below the zygomatic process over the masseter, pierces the buccinator muscle, and opens in the mouth opposite the upper second molar tooth. The place of its opening appears as a minute dark point, often overhung by a little conical elevation. It can be sometimes recognized only by means of a very thin (hair) probe or by the appearance of a jet of saliva.

The *submaxillary gland* lies on the superficial (cutaneous) surface of the mylohyoid muscle, between the lower jaw and the two heads of the digastric muscle; its duct (ductus *Whartonianus*—Whartonian duct) runs, accompanied by a lobe of the gland, over the deep surface of the mylohyoid muscle inwards and forwards, and opens close on the side of lingual frenulum on a little wart-like elevation called the sublingual caruncle (caruncula sublingualis).

The *sublingual gland* lies on the (upper or deep surface) mylohyoid muscle, at the inner side of the jaw, immediately under the mucous membrane of the floor of the mouth. It consists of a conglomeration of glands, and sends a variable number of ducts (ductus Rivini—*Rivini's* duct) upwards, which open either directly along the plica sublingualis or join to form *Bartholini's* duct (ductus Bartholiniani), together with the Whartonian duct, or separately from the latter, on to the caruncle. Sometimes the Bartholinian duct is the common duct of all the salivary glands below the jaw.

The *tonsilla lingualis* is not a real gland.

Blood-vessels.—The *arteries* are derived from the facial artery (arteria facialis submaxillaris externa), which also gives off branches to the cheek and lips. The external carotid also shares in the blood-supply of the tongue; the temporal and internal maxillary arteries, which latter supplies the teeth with arterial blood (arteriae alveolares). The lingual artery from the external carotid (arteria lingualis) supplies the root of the tongue and the floor of the mouth by small (muscular) branches and enters the tongue as the deep lingual artery or ranine artery (arteria profunda linguae), and courses towards the tip of the tongue at the side of the frenulum.

The *veins* empty into the anterior and posterior facial veins, and partially into the internal jugular vein.

Lymph-vessels.—The lymphatic vessels form dense plexuses in the lips and tongue, and join with those of the pharynx to form small canaliculi, which enter chiefly into the submaxillary, cervical, and jugular lymphatic glands.

Nerves.—The *motor nerves* are derived from the hypoglossus, which, running along with the lingual artery, supplies mainly the lingual muscles. The third (submaxillary) division of the trigeminal nerve and the facial nerve also take part in the nerve-supply of the muscles.

The *sensory nerves* come from the lingual nerve (third division of fifth nerve).

The *specific nerves of taste* are derived from the glosso-pharyngeal and lingual nerves and from the chorda tympani (a branch of the facial nerve running within the lingual), which supplies the part not supplied by the glosso-pharyngeal nerve (point and margin of the tongue).

II. PHYSIOLOGY.

The *function* of the oral cavity is of a threefold nature. At one time it forms the first part of the digestive tract, where, so to say, the first act of the digestion is played. The food is masticated and imbued with the secretion of the salivary glands, the saliva, the object of which is to make the morsel slippery, and to convert the starch of the food into dextrin and sugar by the action of the ptyalin.

Further, the mouth contains the chief organ of taste, the tongue, and the terminations of the nerves which serve the sense of taste, *i. e.*, the taste-buds, which are, however, found not only on the point and margin of the tongue and on the hinder part of its dorsum, but also on both sides of the soft palate, on the epiglottis, and inside the larynx. In children the entire dorsum of the tongue serves the sense of taste.

The bulk of the gustatory cups (taste-buds) are found on the papillae, and are most numerous in the walled-in papillae (papillae circumvallatae). On the point of the tongue are the fungiform, and on the margin the foliated, papillae, the carriers of the taste-buds.

Of the gustatory nerves, the glosso-pharyngeal nerve supplies the root of the tongue and soft palate; the chorda tympani, derived from the facial, and running within the lingual nerve, supplies the point and margin. The taste-buds of the epiglottis and larynx are probably supplied by the vagus nerve (nervus vagus). The paths in which the nerves of taste run are very complex, because of the many anastomoses between the glossopharyngeal, trigeminal, and facial nerves, and which are subject to individual variation. Only thus can the many contradictory reports of the various neuroses be explained.

The tastable substances must come in contact with the gustatory cups in order to act as adequate stimuli. We discern generally four kinds of taste: sour, sweet, bitter, and saline, and all other shades of taste can be made up from combinations of these. Nevertheless, as has already been mentioned, smell has a great influence on the sense of taste. The various kinds of taste are, however, not perceived equally by all the gustatory cups; the various papillae, so to say, show special selection, and, indeed, sweet substances are better tasted by the point, and bitter substances better by the base, of the tongue. Mechanical and thermic stimulation are without influence on the organs of taste. On the other hand, one speaks of a so-called "electric taste," which is perceived if an electric current flows through the tongue, viz., a perception of sourness on the anode and bitterness on the kathode (sharpness or alkalinity). The so-called after-taste is to be explained by some particles of the substance remaining on the tongue or papillae, especially on the circumvallate papillae.

Lastly, the mouth forms, together with the pharynx, the resonator for articulate speech and singing. This resonator is altered in its shape by muscular action, in particular by the tongue and soft palate, but also by the lips and cheeks, and is thus very important for the formation of sounds, vowels as well as consonants. It serves also as a mouth-piece, modifying the sounds suitably. In producing consonants, the mouth-piece at one or other part is either shut or narrowed, and the passing of which by the expirated air produces a noise; here the mouth-piece actually forms the sound. In both groups, consonant and vowel speech, the soft palate is drawn up towards the nasal cavity, which, however, is not completely shut off from the mouth; whereas in the production of the so-called nasal consonants or semi-vowels, m, n, and ng, the soft palate hangs down flaccidly, so that the air-column in the nasal cavity vibrates also.

Zarniko points out that all sounds of speech can be produced with perfect purity, even in incomplete closure of the palatine velum, *i. e.*, with nasal resonance. As the use of nasal resonance affords the greatest effect with the least effort, he maintains that speaking and singing with the palatine veil correspondingly opened is the only natural and intentional way. According to *M. Schmidt*, a complete closure is necessary for the production of pure vowels.

The mechanism of swallowing, which only takes place in the mouth in a small measure, will be discussed in the chapter on the Physiology of the Pharynx.

III. METHOD AND COURSE OF EXAMINATION.

A. Method of Examination.—In the examination of a patient's mouth the first thing is inspection. In the second place comes palpation and the detection of any unnatural odour, and in many cases the examination must be completed by chemical and microscopic tests of the secretions and morbid tissues, and by a correct estimation of the patient's sense of taste.

We directly inspect the cavity by either natural or artificial light. The latter is perhaps preferable, and we throw it into the mouth by means of a reflector, using a spatula or tongue depressor for the purpose of keeping down the tongue and pushing away the cheek. In order to inspect the floor of the mouth and under surface of the tongue the patient is directed to open the mouth widely and to put the point of the tongue on the upper teeth. Should the patient be too inexpert, or the tongue be too short and thick, the point can be seized with a clean cloth and held in the position required. The hinder part of the back of the tongue is best viewed by means of a throat mirror.

Palpation with the finger steps in, or completes inspection where we are not able to see, and if we desire to ascertain the consistence or dimensions of certain parts, *i. e.*, tumour, foreign bodies, etc. If possible, palpation should be bimanual—one finger outside and the other inside.

We examine the sense of taste in such a way that a small quantity of tastable matter in solution is brought into contact with the tongue.

B. The Course of Examination.—It is important to inquire as to the history or anamnesis of the case, and as to the subjective symptoms. With respect to the anamnesis, we can refer to what we have said previously in regard to the nose.

The patient may report and complain of trouble in speech, respiration, swallowing, and perhaps secretion, and also of disorders of sensibility and taste.

Disorders of Speech.—Articulation may be affected by reason of inflammatory processes, tumours, and paralysis in the region of the mouth (peripheral), but also through disease of the central nervous system, *e. g.*, disseminated sclerosis, progressive muscular atrophy (bulbar paralysis), etc. Speaking appears difficult, and the speech itself might appear heavy, clumsy, inarticulated, indistinct, interrupted, or retarded.

Disorders of the respiration are seldom due to pathological processes of the cavity of the mouth, and are only produced by excessive swelling of parts at or about the entrance of the larynx.

Swallowing is sometimes difficult in various acute inflammatory processes of the mucous membrane, in particular of the tongue and salivary glands. Indeed, it might be so severe that eating is almost impossible.

Anomalies of the Secretion.—*Increased secretion* (hypersalivation, ptyalism) usually accompanies inflammation of the oral mucous membrane. It is often a symptom in certain forms of poisoning (mercury, iodine, pilocarpine, etc.), or occurs after partaking of various spices; or it follows the introduction of instruments for diagnostic or therapeutic purposes. In other cases it is a reflex symptom, due to divers irritations, as from dyspepsia, intestinal parasites, pregnancy, emotion, and is also often observed in nervous or hysterical persons.

The *paralytic salivation* observed in bulbar paralysis, paralysis agitans, and various psychoses is probably caused by the secretion being uncontrolled, and in some of these cases the salivation is often periodical.

The *diminution of the salivary secretion* occurs after the taking of substances (drugs) which paralyze secretion, *e. g.*, atropine, in incomplete or complete obstruction of the salivary ducts by tumours in the vicinity, salivary calculi; in diseases causing specific deviation of body fluids, as in high fever, diabetes, chronic Bright's disease, enteritis, etc. Old people and neurasthenic and hysterical persons often complain of dryness of the mouth (xerostomia), a consequence of the diminished secretion.

Disorders of sensibility and taste are often complained of, especially by nervous patients, *e. g.*, as of suffering pain while speaking, smoking, or eating. Or there are complaints of certain paraesthesiae (tingling, burning, numbness, etc.), mostly at the point of the tongue.

Examination begins with the inspection of the lips, which are gently separated from each other; then of the teeth, gums, hard palate (the head being well reclined); further, both pockets between the teeth and cheek and the cheek itself, and last, but not least, the tongue. At the same time any particular odor must be noticed.

In the majority of cases fetor ex ore can be explained by carious teeth or decomposed remains of the food between or in hollow teeth, or from tartar, ulcerations, and injuries, etc. If one cannot find the cause of the bad smell in the mouth, the tonsils or the nose must be examined. In a few cases bronchitis foetida may be discovered.

On examining the tongue, attention should be given to the color, which essentially depends on the state of health. Ascertain whether the dorsum of the tongue *be very much furred,* especially if the hinder part appears white, yellow, or greenish. The shade of the tongue is often influenced by the food passing over it; therefore it is always well to inquire concerning the food, drinks, or drugs taken prior to the examination, in order to avoid mistakes.

Some people, such as smokers or drinkers, always have a furred tongue. This film (fur) consists mainly of microörganisms, epithelial cells, and débris. The tongue also shows normally much unevenness. If it is much furrowed (sometimes a congenital abnormality), one speaks of a lingua dissectata. On its margin the impressions of the teeth may often be seen. On the other hand, defects, changes in the surface by scars, by inflammatory processes, *e. g.*, in syphilis, cancer, lupus, cysts, naevi, etc., may be observed.

IV. HYGIENE AND PROPHYLAXIS.

The microörganisms which enter the mouth during respiration and with the food are a constant cause of decomposition and putrefaction in the region of the teeth. The fur coat of the tongue, which contains so many microbes, also plays an important rôle, which is often not sufficiently recognized. We should, therefore, insist strongly on a regular cleansing of the mouth and teeth, particularly with respect to the remaining particles of food, which are such a good nidus for the development and increase of bacteria.

That not the most scrupulous purification of the mouth would be sufficient to sterilize it, even for a short time, we need not especially emphasize. We can only, by a thorough cleansing, check the further development of the microörganisms within very modest limits.

The mouth should be cleansed at least every morning and evening, and also after meals, and this is done by first removing all remains of food with a goose-quill toothpick or by a waxed dental thread; secondly, by washing the mouth and throat with tepid water, to which is added a little cooking salt; thirdly, by cleansing the teeth in front and back, above and below, by a tooth-brush, with dental soap or powder, made up of chalk and carbonated magnesia; fourthly, by cleansing the back of the tongue with a scraper; and fifthly, by repeated washings as in number two. It is taken for granted that all the appliances should be kept clean.

Whether the mouth-wash is flavored with aromatic essences instead of using simple salt solution is really only a matter of taste, but we will not deny that they have a refreshing and deodorizing effect. We should desire, however, to warn as to the use of too strong disinfectants, because of their corrosive action. Disinfectant mouth-washes are only to be used in special diseases and not unless ordered by the physician.

Artificial teeth should be taken out during the night and put in salt solution, otherwise they should be treated as are the natural teeth. For details with regard to the care of the teeth see text-books on odontology.

Children should be trained from early youth as to the proper cleansing of the teeth and mouth. The milk teeth must be taken care of in just the same way as the permanent teeth. In the suckling period it is best not to interfere too much with the mouth if there is no need for it.

SPECIAL SECTION.

I. MALFORMATIONS AND DEFORMITIES.

Etiology and Pathology.—Malformations and deformities are either congenital or acquired. The first are due to disturbance of development in the embryo, and the latter are caused by injuries, ulceration, and the retraction of scars.

(*a*) *In the Lips.*—The cleft-lip or hare-lip (labium leporinum), as it is called, is a very common occurrence. It is found oftener on the left than on the right side, and is more often unilateral than bilateral. It is sometimes indicated by only a slight notch; in other cases again it extends to the nose and is combined with a cleft jaw and cleft palate. Hare-lip is always congenital, but occasionally the lip is divided by a cut or thrust-wound.

Of other anomalies, there are: *Hypertrophy* (*elephantiasis; macrocheilia*) of the upper lip is due to hypertrophy of the labial glands or to chronic infiltration from repeated nasal catarrhs; as we see it so often in scrofulosis, especially in the colder season. The lip is, in these cases, often cracked, fissured, and everted. The reverse is the case in children, who are accustomed to breathe through the mouth, and whose upper lips are small and shortened, with the lower lip everted.

The double lip occurs when the upper lip is divided into two more or less broad parts by a deep fissure.

Entire *absence of lips* (*acheilia*), *abnormal smallness* (*microcheilia*), and lastly *adhesions of the lips* to each other or to the gum (*syncheilia*, and *synechia*), are also occasionally encountered.

(*b*) *In the Palate.*—Cleft-palate (palatum fissum) is either limited to the soft or to the hard palate, or it extends throughout both. The cleft is mostly single and on the left side of the vomer, which in such a case shows deviation towards the right-hand side; in the soft palate the cleft always runs in the median line. If the cleft is double, *e. g.*, on both sides of the vomer, it is spoken of as *wolf's throat.*

The vault of the hard palate may very often deviate from the normal. An abnormally high-vaulted palate is supposed to be a stigma of degeneration, and is seen in association with many mental diseases (epilepsy); but it is also seen in children who breathe through the mouth, the nasal respiration being obstructed by adenoid growths. In these latter a high-vaulted palate is often seen, and is then associated with abnormal position of the teeth.

(*c*) *In the Tongue.*—Congenital abnormalities are rare. An abnormally large tongue (macroglossia, protrusion of the tongue), due to a congenital enlargement of the lymph-vessels (lymphoma), may be seen in idiots. The opposite condition, a tongue of such very small size as to be almost entirely absent (microglossia), has also been observed. Adhesions of the tongue are mostly acquired. Of more importance, though often exaggerated, is an abnormally long or short frenulum.

(*d*) *In the Jaw.*—Congenital malformations are very rare, and if they occur, are always part of a general malformation of the skull. The jaw-cleft may be mentioned as running between the second incisor and the canine tooth, and is always associated with other abnormalities. Of the acquired deformities, there is the *fibrous ankylosis*, which is caused by fibrous adhesions due to ulcerative processes on the inside of the cheek and in the vicinity of the joint; this fibrous ankylosis (ankylosis spuria) is to be distinguished from true (bony or fibrous) ankylosis in the joint itself.

Symptoms.—The symptoms vary much according to the seat and nature of the anomaly, and the most important symptom is difficulty of speech. Cleft-palate renders feeding difficult, and especially suckling, as the fluid reaches the nose, from which it bubbles forth. An abnormally long frenulum is sometimes accused of being the cause of faulty swallowing; the tongue during the act of suckling is drawn back and upwards and is said to cause choking by depressing the epiglottis. Abnormally short or rigid frenulum (ankyloglossum) may hinder suckling and speaking, for the point of the tongue cannot be raised above the lower teeth.

Diagnosis.—Is generally easy.

Prognosis.—The prognosis depends on the degree, cause, and nature of the deformity, and on the possibility of remedying them by operation.

Treatment.—The frenulum can be severed—which should be done only in stringent cases—in such a way that it is seized between the fore and middle finger of the left hand, the dorsum of the hand being directed upwards, or it can be pushed onto the notch of the handle of a probe and then cut through with *Cooper's* scissors, the curved blades being directed downwards, in order to avoid injuring the ranine artery. The other malformations require osteoplastic operations (see text-books on surgery). It is taken for granted that accompanying maladies, such as rhinitis, adenoids, etc., must be properly treated.

II. INFLAMMATIONS.

ACUTE STOMATITIS (STOMATITIS ACUTA).

The classification of the acute, and—as we would like to point out at once—the chronic inflammations of the oral cavity, meets with considerable difficulty; as the clinical picture is such a diverse one, and changes its character so quickly and often by affecting first one, then another, section of the mouth, or running now more superficially, then more deeply, into the tissues. The classification carried out in the following pages, though not free of all objections, will, I hope, practically satisfy the need for lucidity.

1. STOMATITIS ACUTA CATARRHALIS (ACUTE CATARRH OF THE MOUTH).

Etiology.—Acute inflammation of the mouth is due to mechanical, chemical, and thermic irritations, which are "cutting" of teeth, carious teeth, too hot or too cold food, too spicy or too harsh food, spirits, tobacco, caustics, and other drugs. In infants the use of dirty "comforters," the want of cleanliness, and also the overanxious cleaning of the mouth may cause inflammation of the delicate mucous membrane. Secondary catarrhal stomatitis is found as a constant accompaniment of the acute infectious diseases (see p. 203), in indigestion, in poisoning from some drugs, *e. g.*, iodin, lead, and mercurial treatment, when it is encouraged by individual idiosyncrasy and by uncleanliness with regard to the mouth. It is often in these latter cases, especially in mercurial stomatitis, that the morbid process spreads into the deeper tissue and often assumes an ulcerative character.

Symptoms.—The patient complains of discomfort, which especially occurs while eating solid or warm food; and for this reason small children often cry and refuse the breast or bottle. The tongue is furred and taste diminished; salivation is increased, or the mucous membrane appears dry or covered with a viscous secretion. Other symptoms which may occur are those caused by the various primary diseases.

On examination one finds the mucous membrane affected either in its whole extent, or only the gums (gingivitis); but seldom the tongue alone. The mucous membrane is swollen, red, and covered diffusely or in isolated sections and tracts with mucus. The tongue is usually coated on its dorsum with a yellowish or whitish fur, whereas the point contrasts conspicuously by its bright red color. On the margins of the tongue and on the cheek the impressions caused by the teeth are very distinct.

Sometimes the root of the tongue is the part more affected, and there the lingual tonsils might become diseased. The process is known as tonsillitis sive angina prae-epiglottica; and with all the symptoms and characters of follicular angina, similar to those found in follicular inflammations of the palatine tonsils.

Both often occur in conjunction, but as the symptoms caused by the latter are usually predominant, the inflammation of the lingual tonsil is mostly overlooked (see Part III, page 262).

Prognosis.—The prognosis must be estimated according to the determining cause. Slight inflammation usually subsides after a short time, with or without treatment, but other cases show a greater persistence, and it might become a serious matter for sucklings, who, on account of the pain, will refuse food.

Treatment.—It is essential to remove or prevent all irritation which is apt to keep up or to renew the inflammation. The food, at least in the beginning of the disease, should consist of cold fluid or pap (milk, barley-water, cold soup, eggs, jam, gruel, rice-milk, etc.); and infants must be fed by spoon on cold milk.

Local treatment ought not to be exaggerated, for all the cleanings and paintings only cause unpleasant results. In slight cases dietary prescriptions and a mouth-wash consisting of a tepid infusion of camomile, to which a teaspoon of *Burow's* solution may be added, is sufficient; or a mouth-wash of tincture of myrrh (25 drops to ½ litre) can be used. I usually prescribe, in cases of severe pain, the following mouth-wash:

℞. Tinct. myrrha.
Tinct. rhatan .... aa 10.00
Tinct. thebain. 5.00
Sig.—To make a mouth-wash, add 25 drops to a tumbler of cold water.

The mouth-wash should not be used as a gargle, but only as a mouth-wash, *i. e.*, a quantity taken into the mouth and then moved to and fro within the closed cavity by slightly blowing out the cheeks. In infants it is best to cleanse the mouth cautiously with a clean swab dipped in a weak solution of borax (1:10).

In adults it might be necessary to paint the mucous membrane with silver nitrate (0.5:100).

2. PHLEGMONOUS STOMATITIS.

Etiology.—Phlegmonous inflammation often affects one or other part of the mucous membrane, and the most common site for the phlegmon is the tongue.

Among the causes, injuries by fish-bones, bites, stings, etc., followed by infection from septic or infectious germs, play the chief part. Sometimes constitutional disease (dyscrasic ulcers) form the basis of a severe phlegmonous process, or the latter is only a symptom of severe general disease, *e. g.*, scarlet-fever, small-pox, typhoid, anthrax, etc.

Symptoms.—After stings from insects, burning or scalding often appears in a very short time, and the parts concerned become swollen and enormously infiltrated, which, however, soon subsides spontaneously or under proper treatment, and seldom goes on to abscess formation. Purulent inflammation is almost always unilateral, and most commonly affects the root of the tongue and the lingual tonsil; the abscess is of the size of a pea, but may assume larger proportions.

In such a case the tongue is so swollen that it does not find room enough in the mouth and may protrude; it is moved with difficulty or is entirely amobile, and is always thickly furred. At the same time there is violent pain on speaking and swallowing; great salivation; the regional lymphatic glands are swollen; and if the aditus ad laryngem is implicated, there is difficulty in breathing, especially at night (in the reclining position).

Diagnosis.—The diagnosis is made from the symptoms, inspection, and, if suppuration takes place, by palpation. The abscess is not always easy to be felt, because of its deep-seated position and the tenderness and tension of the affected parts; but it is just the tenderness which will guide us in making our diagnosis.

Prognosis.—The prognosis in simple phlegmon is favorable, but if the disease has extended over a large area or if suppuration has taken place, there is danger of asphyxia or sepsis and pyaemia.

Treatment.—At the commencement the diet must be regulated (cold fluid food), and applications of ice, as well as the swallowing of small pieces of ice, and leeches to the angle of the jaw, must be applied. Should this treatment prove insufficient, the inflammation must be accelerated by hot-water fomentation; a mouth-wash must be used (infusion of camomile to which a teaspoon of *Burow's* solution is added); and where fluctuation is found or on the site of the greatest tenderness, an incision must be made. Severe hemorrhage is stopped by plugging. For the after-treatment, thorough cleansing of the mouth is very important. Sometimes a certain degree of induration of the tongue remains.

3. STOMATITIS EXSUDATIVA.

Exudative stomatitis is characterized by the formation of sero-purulent or haemorrhagic vesicles or bullae, and in other cases by a fibrinous exudation. We find an eruption of vesicles in herpes and pemphigus, after scalding or burning, in erysipelas, foot-and-mouth disease, variola, and varicella; to this class also belongs urticaria, which occurs on the mucous membrane of the mouth. Fibrinous exudation characterizes stomatitis aphthosa (aphthae).

(a) Herpes labialis et buccalis (Stomatitis Herpetica; Herpes of the Lip or Cheek).—*Etiology.*—Among the causes enumerated are intestinal disorders, some acute infections, as coryza, influenza, pneumonia, epidemic cerebrospinal meningitis, etc. (compare Part III, p. 272).

Symptoms.—The vesicles of herpes are of the size of the head of a pin to a pea, and occur mostly on the lips, often in association with others, on the facial skin, and sometimes also on the inside of the cheek, on the hard palate, or on the tongue. The eruption always occurs in clusters, and is often followed by febrile symptoms. There are often several attacks, and at first the contents of the vesicles are clear and transparent, but later they become turbid, and the vesicles tend to dry up after a few days and leave little crusts or scabs which, if removed, are renewed. In the mucous membrane of the mouth the vesicles nearly always burst so early that one only finds in their place small circular, at first blood-stained, and later on yellowish, excoriations, which are surrounded by a well-marked red zone. Here also the eruptions are arranged in groups, and are always limited to one side only (hemiglossitis herpetica—one-sided herpetiform glossitis). It is probable that this disease is, like herpes zoster of the skin, a trophoneurosis in the region supplied by the trigeminal nerve.

The subjective symptoms in herpes labialis are insignificant, and there might be a feeling of tension or heat. In herpes of the mucous membrane, on the other hand, *pain,* especially on eating, might be very distressing.

Course.—The course usually lasts from a few days to a few weeks. Some chronic forms have been described which relapse from time to time.

(b) **Pemphigus.**—*Etiology.*—Whether pemphigus is a trophoneurosis like herpes zoster or whether it is an infectious disease is not yet certain.

Symptoms.—It occurs mostly in middle-aged or elderly people. The formation of vesicles or bullae often escapes observation, as in the previous disease. One sees on the soft palate or cheek, but not so frequently on the tongue, large, well-defined, gray or yellowish excoriations which still show the remains of the burst epidermis, and which heal without scar. Swallowing is very painful, and there is salivation and an abominable foetor. The disease shows great inclination to relapse, and by this circumstance elderly people may become so weak that they easily succumb to an intercurrent malady.

(c) **Aphthae (Stomatitis aphthosa seu fibrinosa; Aphthous Stomatitis).**—*Etiology.*—The origin of aphthae must be traced to infection. This is proved by its contagiousness, for not only several children, but also the adults of the same family, may be affected by the disease. Children are very liable to contract it, especially during dentition, between the first and third year. It is fostered by want of cleanliness, gastric disorders, febrile diseases, etc.

Symptoms.—It occurs with febrile symptoms on various places in the mouth, such as in the vestibulum, in the pockets of the cheek, and on the floor of the mouth. Spots or plaques are seen which, at first vividly red, are later on somewhat whitish or yellowish, and are surrounded by a red area of infiltration. In some cases these spots or plaques are scanty; in others, they are very numerous, and multiply themselves to form several crops, and have also a tendency to unite. In some cases the more posterior portions of the mouth and the tonsils are affected.

The eruptions do not form vesicles, but are from the first onset fibrinous exudations which exist in the epithelium itself.

During the first three days the aphthae increase, and are accompanied by ample salivation and considerable pain; the submaxillary glands are swollen, and there is foetor ex ore. Then the injection of the mucous membrane subsides, the exudate shrinks and is discharged, leaving red spots behind which gradually disappear. There is no formation of scars. The whole process may be very severe and spread over so wide an area that the little patients are liable to become debilitated during its course.

(d) **Foot-and-mouth Disease (Epidemic Aphthous Stomatitis; Maul- und Klauenseuche).**—*Etiology.*—The disease is epizootic, and occurs in cattle, cows, sheep, pigs, and horses, whence it can be transmitted to man through the milk or by contagion. The specific germ has not yet been discovered.

Symptoms.—After a stage of incubation, lasting from five to ten days, the disease begins with fever, often with a rigor; the patient feels very weak, giddy, and complains of pains and aches in the back and stomach; of nausea, anorexia, etc. Small yellowish vesicles the size of a hemp-seed are formed on the lips, gums, or tongue, seldom on the hard palate or posterior pharynx. The contents of the vesicles are at first quite clear, but later on become turbid. The vesicles soon burst, leaving behind dusky erosions or superficial ulcers. The whole mucous membrane is swollen, there is salivation, and eating is rendered very painful. In many cases a vesicular eruption also appears on the external skin, which sometimes shows a purpuric character.

The disease mostly runs a course of several weeks, ending in complete recovery, but death has occurred in some cases.

Diagnosis.—The diagnosis of the various forms of exudative stomatitis, and especially the differential diagnosis, is not always easy, for the efflorescences show a most varied clinical picture. Special difficulties not seldom arise in making a distinction between the herpetic and aphthous forms of stomatitis, and sometimes, if the history is doubtful, between these two diseases and epidemic stomatitis. Syphilis is not often to be confounded with them, as the acute course of the exudative process alone would render an error improbable. Sometimes in diphtheria similar symptoms occur, but here a bacteriological examination on the one hand, and, on the other hand, some typical herpetic or aphthous plaques which are never wanting, will insure the diagnosis. In certain cases similar eruptions on the external skin will serve as a good guide as to the nature of the disease.

Treatment.—With regard to treatment, one chief requirement is indispensable, *i. e.*, scrupulous cleanliness of the mouth; in adults, by disinfectant mouth-washing (aluminum acetate, peroxide of hydrogen, permanganate of potash), and in children who cannot gargle or clean their mouths, by painting or brushing with borax, 5, to glycerine, 25, or chinosol or chinolin, 0.4, spir. vini et glycerin, āā 10. For the painting, a camel's-hair brush can be used. *Heubner* advises a 3 per cent. solution of carbolic acid; the brush is moistened with the solution so as not to drip, and then each single spot is touched with it. In more severe cases a 2 to 10 per cent. solution of silver nitrate or solid lapis infernalis may be used. In severe pain, painting with cocain or alypin, 5 to 10 per cent., is useful, in order to render eating less painful. For the rest we refer to what has already been said with regard to catarrhal stomatitis.

III. STOMATITIS CHRONICA (CHRONIC STOMATITIS).

1. STOMATITIS CHRONICA CATARRHALIS (CHRONIC CATARRH OF THE MOUTH).

Etiology.—The chronic form of catarrh in the mouth is often found in abusers of alcohol and tobacco, with carious teeth and unclean habits. Sometimes the inflammation is limited to the gums (in pregnancy), which protrude between the teeth.

Symptoms.—Save from disturbances of taste and salivation, there is very little cause for complaint. The mucous membrane on the back of the tongue or on the cheek is infiltrated, slimy, and here and there excoriated and atrophied, and the vessels are much dilated.

A variety of this chronic catarrh is that form described by *Schech* as stomatitis sicca (dry stomatitis), and is found only in older or uraemic persons. Here the mucous membrane is much injected, shiny, and covered with a glassy or viscous secretion. The patients complain of unpleasant dryness in the mouth or throat, of difficulty in speaking and swallowing. The disease is not to be confounded with xerostoma, where inflammation is wanting or absent. (See p. 188.)

Prognosis.—The prognosis is favorable, but the course may take years, if the causes cannot be removed.

Treatment.—Must be eminently causal. The local treatment, to a certain degree, is the same as in the acute catarrh. As the mucous membrane is often to a large extent insensitive, painting with silver nitrate or lapis infernalis is often beneficial. Great benefit also accrues from the use of certain mineral waters (Salzbrunn, Vichy, Ems, Kissingen, etc.).

2. GLOSSITIS CHRONICA SUPERFICIALIS.

(a) Leucoplakia Oris (Psoriasis sive Ichthyosis Oris; Leucoplakia; Buccal Psoriasis).—*Etiology.*—Chronic superficial inflammation of the mucous membrane is produced by continued irritation, especially by excessive smoking; that is why we so often see it in men. Local (such as an abnormal delicacy of the mucous membrane) or general predisposition (syphilis) contributes much to the occurrence. Leucoplakia is not necessarily a symptom of syphilis. This is proved by persons who never had syphilis showing white patches in the mouth; and, further, that in some cases mercurial inunctions were of little avail, and that, following upon long-continued mercurial treatment, such leucoplaques (leucomas) occur, which disappear, however, but show a distinct inclination to relapse, in opposition to the primary leucoplaques, which are very resistant and not amenable to treatment. On no account refer these leucoplaques to an existing syphilis, for they are not a symptom of it, *but the sequelae of antisyphilitic treatment.* Where such white patches are found without other signs of syphilis, it would be a great mistake to submit the patient to mercurial treatment, for it would only aggravate the evil.

Symptoms.—The tongue usually, sometimes also the lips and cheek, are covered with white or bluish patches, of various sizes and irregular in shape; sometimes sharply defined, in other cases diffusely passing over into normal mucous membrane, often divided into several patches by fissures or furrows. In some cases one finds islands of normal red mucous membrane enclosed in such fields of white patches, especially on the tongue. In long-standing cases these white patches are thickened, elevated, uneven, or cracked.

The patients do not usually complain if the disease has not spread far, and even in some advanced and long-standing cases the subjective symptoms are relatively slight. There is a sensation of dryness or roughness in the mouth, speaking, swallowing, and chewing are rendered uncomfortable, and hot and spiced food, particularly, is most disliked; but the whole mouth becomes in time very sensitive, and if the mucous membrane becomes fissured or in places loses its epithelium, the symptoms are aggravated.

Diagnosis.—The discrimination from syphilis is not always easy. If the white patches show marked resistance, increasing and altering very slowly, we then diagnose leucoplakia; whereas syphilitic patches are characterized by the papules appearing and disappearing relatively quickly. There will also be other signs of syphilis, and mercurial treatment will soon settle the question. Syphilitic plaques very seldom leave scars behind (*Erb's* syphilitic scars, p. 208), but leucoplakia nearly always does.

Besides syphilis, in other cases, *lichen planus* must be considered. In this disease, also, white patches appear on the mucous membrane of the mouth. These patches are, for the most part, much smaller, more silvery, shinier, are raised and distinctly arranged in stripes or striae. In the skin we will probably not fail to discover the same eruption of lichen ruber planus, which, however, soon subsides under the influence of arsenic.

Prognosis.—Leucoplakia is always a very tedious disease, and takes years to heal. Prognosis is, therefore, somewhat unfavorable, and might become serious because of its predisposition to the growth of epithelioma, though this liability is often much overrated.

An epithelioma growing on the site of a leucoplakia is said to be much less malignant than other carcinomatous tumors in the region of the mouth. According to *Reclus,* this is due to the mucosa assuming in leucoplakia all the characteristics of the skin, and the cancroids of which, as is known, are far less malignant.

Treatment.—It is most essential to prevent any further irritation of the mucous membrane. Smoking, drinking, and mercurial treatment must be discontinued, and all carious teeth must be extracted or put in order. Local treatment is not very successful; though painting with lactic acid (20 to 50 per cent.) or with a mixture of salicylic acid, 1.00, spirit of wine, 5.00, glycerin, 10.00 (*Beregszászy's* mixture), will perhaps be useful. The painting is to be done every two to three days, and in the meanwhile the mouth should be washed or gargled with peroxide of hydrogen (a teaspoonful to a glass of water). Equally good are weak solutions of silver nitrate (5 to 10 per cent.) or chromic acid (1 to 3 per cent.).* Stronger solutions are not advisable, for they not only aggravate the evil, but foster the predisposition to a subsequent cancer.

(b) Nigrities linguae (Lingua Nigra; Black Tongue; Hairy Tongue).—*Etiology.*—Like leucoplakia, the disease now to be described is also caused by a superficial inflammation of the tongue. The superficial layers of the epithelium and the filiform papillae hypertrophy and become corneous and assume a yellow or brown color (*Shech*). The process is that of keratosis.

Other writers look upon it not as a keratosis, but seek to explain it as a pigmentary degeneration of mycotic origin.

Symptoms.—There are usually no complaints on the part of the patients, or they may remark on a feeling of dryness, hairiness, sometimes in combination, with diminution of taste and an evil smell from the mouth. If one examines the tongue, it will be found thickly coated with a dusky brown or black fur, which is nothing else than the hair-like hypertrophied corneous and pigmented filiform papillae. By scraping with a spatula, from before backwards, the papillae can be made to sit up like fine spines, so that the back of the tongue has an appearance similar to the tongue of a cat.

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*Translating Editor's footnote: Painting the sites with Liq. Plumbi Subacetatis Fort.. and then rinsing the mouth at once has been found most efficacious in the editor's experience. This treatment has proved useful in cases of smoker's irritable, fine, multiple papillomata of the hard palate, the result of persistent pipe smoking.—F. W. F. R.

Diagnosis.—Mistakes are possible only in artificial discoloration, as may occur after the taking of claret, certain drugs (iron) or fruits, or chocolate; or even from the licking of ink.

Prognosis.—Good, though the disease is very tedious and obdurate.

Treatment.—Careful attention to the mouth is a matter of course. *Unna* recommends local painting of the discolored portions with salicylated or resorcinated ether (resorcin, 10.00; collodion, 5.00; ether, 100.00), and afterwards sponging with peroxide of hydrogen. Caustics or instrumental removal should only be employed in very serious cases.

If the discoloration is insignificant, and the discomfort of the patient slight, it is best to leave things alone.

(c) **Lingua geographica (exfoliatio linguae areata) (Geographical Tongue; Annulus Migrans).**—*Etiology and Pathology.*—This affection is characterized by the appearance of circular excoriations of the mucous membrane. Its cause is not yet known: probably it is due to a superficial inflammation. Its appearance closely resembles syphilitic plaques, but it has no connection whatever with syphilis. Anaemic or dyspeptic conditions are supposed to play a predisposing part; and dentition also is said to predispose to these "idiopathic plaques" of the mucous membrane, for they are mostly met with in young children.

Symptoms.—The tongue shows red patches surrounded by a yellowish or whitish yellow margin of doubled contour. These patches occur on various places, and are sometimes here, and sometimes there, and this quick "migration" is characteristic of the disease. Patients suffer little; sometimes there exists an intolerance of acid and spiced foods, and tobacco, spirits, etc., in older persons.

Diagnosis.—Syphilis is most likely to be mistaken for it. The exfoliations are localized only to the tongue, whereas syphilitic plaques are seldom limited to the tongue alone, and are usually also found on other places in the mouth (soft palate, tonsils, lips, cheeks). The "migration" of the plaques is pathognomonic.

Prognosis.—The disease is not a serious one, but is very obstinate.

Treatment is of little avail. *Unna* recommends a special mouth-wash:

℞.	Aq. sub. sulphurosa.	
	Aq. menth. pip.	āā 100.00
	Flor. sulph.	
	Syr. simpl.	āā 20.00
	Tragacanth	2.00

To make a mouth-wash, to be shaken before use.

The patient is ordered to cleanse the mouth thoroughly three times a day for five minutes. Small children are painted with borax or chinolin.

℞.	Sodii biborac	5.00
	Glycerin	25.00

Or—

Chinolin	0.4
Glycerini	
Spirit. vini	āā 10.00

(d) **Moeller's Glossitis Superficialis.**—This affection, called after its first observer, is, like the geographical tongue, characterized by chronic excoriations, which, however, are not surrounded by a double contoured margin, but show an evenly distributed redness and remain stationary in their primary place or site of origin, in contradistinction to those on the geographical tongue, where the exfoliations quickly change from place to place. The disease occurs in adults only, and is very painful, whereas lingua geographica is found chiefly in children, without causing any pain.

Treatment.—The same as in chronic catarrhal stomatitis.

3. GLOSSITIS CHRONICA PARENCHYMATOSA (PARENCHYMATOUS GLOSSITIS; MACROGLOSSIA; PROLAPSUS LINGUAE).

Etiology and Pathology.—Macroglossia is often congenital, as already mentioned (p. 181). In rare cases it is acquired, and is then the consequence of oft-repeated inflammation of the tongue. (See Fig. 65.)

Symptoms.—The enlargement of the tongue, which may attain great proportions, is liable to impede speaking, eating, and even respiration, and is always accompanied by copious salivation. The tongue does not find enough room in the mouth, and so protrudes and hangs down. This causes the part exposed to become desiccated and cracked. The teeth are pushed forward and are liable to fall out. In a more localized affection, the tongue shows at single places tuberous thickening, which causes merely slight difficulties in its motility.

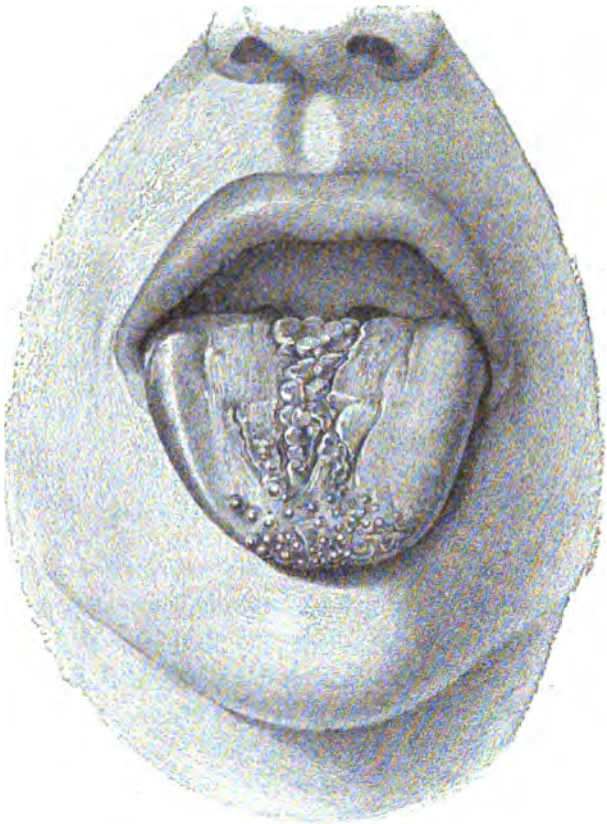

Fig. 65.—Macroglossia (after *Hochenegg*).

Treatment.—This consists in excision of a wedge-shaped portion or in the amputation of the tongue by the galvano-cautery. Ligature of the lingual artery has been advised by *Fehleisen*.

Circumscribed, well-defined, nodular enlargements may be sometimes successfully treated by painting with tincture of iodin or by cauterization with solid silver nitrate.

IV. STOMATITIS ULCEROSA (CHRONIC ULCERATION OF THE TONGUE).

Ulceration of the tongue occurs in various forms:

(*a*) *Idiopathic ulcer* shows a localised character, the clinical features changing with the different causes or microbes. To this class belong the idiopathic ulcer (stomacaca), the stomatitis ulceromembranosa (angina of *Plaut-Vincent*), and the aphthae of *Bednar*.

(*b*) *Symptomatic Ulcer.*—Here the affection of the mouth is only a part of a general diseased condition, such as scurvy, diabetes, leukaemia. (Diphtheria and syphilis, which also produce ulceration in the mouth, will be discussed later on.) It occurs subsequently to a general cachexia, the mouth being the locus minoris resistentiae; lastly, following on the poisoning from various drugs, *e. g.*, mercury, bismuth, lead, arsenic, etc.

(*c*) *Traumatic ulcer, e. g.*, from whooping-cough or from bad teeth.

(a) STOMACACA (ULCERATION OF THE MOUTH).

Etiology.—Idiopathic ulcer never occurs in the mouth of sucklings or of toothless old people; this circumstance indicates that the teeth (caries) play a part in the disease. It is met with most commonly in children, but adults also are not immune from it. Sometimes it has been found epidemic (in barracks), and this leads to the opinion that the disease may probably be contagious, which has been certified in various cases by bacteriological investigations.

Until now no specific germ has been discovered. It is rather more probable that the disease is the effect of several saprophytic microörganisms of putrefaction which are always present in the mouth.

Bad hygienic conditions, and debility due to illness or convalescence after infectious diseases are the predisposing factors.

Symptoms and Course.—The disease begins with an acute onset, mostly near carious teeth. The gum swells, becomes discolored, and bleeds on being touched. After a few days, or rarely after a few weeks, it shows a greyish-yellow patch, covered with a cheesy, evil-smelling coat, which soon ulcerates. The teeth loosen, fall out, or are removed without pain. There is ptyalism and swelling of the submaxillary glands. Feeding and speaking are painful. The ulceration frequently spreads into the mucous membrane of the lips and cheek, and in neglected cases may lead to gangrenous destruction, which may even affect the bones. If the process is so far advanced, general sepsis develops and finally terminates in death. Usually, however, the general health is not much affected save from the frequently severe pain.

Diagnosis.—It may be confounded with scurvy and mercurial stomatitis, especially at the beginning. Stomacaca is nearly always a local affection, whereas scurvy is a constitutional disease and never shows greyish-yellow patches on the gum. Mercurial stomatitis, which may show the same clinical picture, is determined by the previous history.

Prognosis.—If the disease is properly treated, the prognosis is always good.

Treatment.—The treatment follows the same lines as in catarrhal stomatitis. *Heubner* maintains that cauterizing the ulcerated parts with pure carbolic acid is very effective. He uses a glass rod for this purpose. We prefer to recommend as a disinfectant and deodorant formic aldehyde, especially in the form of formamint tabloids. Formamint is a compound of sugar of milk and formic aldehyde, of which several tablets are taken daily. Chlorate of potassium, which is still in use, should not be given, on account of its danger. It is a matter of course that the mouth and teeth be regularly cleaned.

Attention should be directed to the hygienic conditions and to the general health by strengthening the body, with concentrated food, such as eggs with sugar and wine, coffee with milk and sugar, etc.

(b) STOMATITIS ULCERO-MEMBRANOSA (ANGINA OF PLAUT-VINCENT).

Etiology and Pathology.—This disease, which has recently occupied the attention of the medical world much, is an affection of the mouth and throat caused by certain spindle-shaped or fusiform bacilli, first described by *Plaut* and *Vincent,* and by spirochaetae; the latter, however, play only a minor part in causation. Both microörganisms are occasionally found as saprophytes on the healthy mucous membrane of the mouth, but may become pathogenic after long illness, general or local affections, carious teeth, etc. There is no agreement as to the contagious nature of the disease, though epidemics have occurred.

According to *Vincent,* the disease occurs at any age and among all classes of people; seldom, however, after the thirty-fifth year.

Symptoms.—The affection is characterized by the formation of ulcers and membranes, which in the one form are more numerous on the mucous membrane of the mouth, near carious teeth, and in other forms are localized more to the soft palate or tonsils, spreading sometimes down to the larynx.

The ulcers show an irregular, hard, crater-shaped edge, and with the tendency to spread into the deeper tissues more than along the surface. The adjacent mucous membrane is inflamed and swollen. There is salivation, foetor, and swelling of the submaxillary glands.

The affection is mostly limited to one side, and does not much affect the general health. In some cases complications of the kidneys have been observed.

Diagnosis.—The disease must be distinguished from diphtheria and syphilis. This can in any case be done by the microscopic discovery of the fusiform bacilli and spirochaetae in the cheesy detritus taken from the ulcers and stained with carbolfuchsin.

Prognosis.—Is usually good, but the course is, in contradistinction to stomacaca, very tedious and obstinate.

Treatment.—Carious teeth must be removed, and the mouth and throat cleansed and disinfected by mouth-washes or gargles or tablets of formamint. Eventually, caustics (silver nitrate or chromic acid) have to be used. (See p. 40.) The patient may be isolated in order to prevent contagion.

(c) BEDNAR'S APHTHAE.

Etiology and Pathology.—The disease is only observed in infants. All agree in that it is caused by damaging or injuring the delicate mucous membrane by rough or unskilful wiping of the little one's mouth. *The hinder and more lateral parts of the hard palate,* where the mucous membrane is firmly attached to its base, are always and exclusively affected. The excoriations then form the path of entrance of the staphylococci.

Symptoms.—The mucous membrane on both sides of the median raphé and in front of the attachment of the soft palate shows symmetrically small, flat ulcers, which appear like small yellow patches, rounded or oval in shape, with red and well-defined edges.

The affection causes little or no disturbance of health, save in the rare cases where the disease also invades the soft palate or tonsils, leading to septic infection, or is complicated by thrush or diphtheria.

Diagnosis.—Is founded on the seat of the ulcers and on the age of the patient.

Treatment.—It is often only sufficient to forbid wiping the mouth, when the affection will heal up within a few days. In other cases light and careful painting with 1 to 2 per cent. nitrate of silver may be tried. As there is no danger of contagion, breast-fed children need not be weaned.

(d) STOMATITIS SCORBUTICA (SCURVY) (SCHARBOCK).

Etiology.—Scurvy, which is not often observed at the present day, is probably an infectious disease, the outbreak of which is encouraged by bad hygienic conditions and particularly by feeding on salted meat or impure food.

Symptoms.—After the prodromal stage (languor, oppression, palpitation, excruciating pain in the legs, fever, etc.), ecchymoses in the skin and mucous membrane occur, and simultaneously the gums become affected; they become swollen, painful, and discolored. The parts between the teeth especially swell so much that they protrude like pads, bleeding freely on being touched. Curiously enough, any gaps between the teeth are not affected. In severe cases the gums become ulcerated, the ulcers bleeding freely, showing a shiny, evil-smelling film; the teeth loosen and fall out, and the adjacent mucous membrane is often implicated, so that the disease conveys the impression of an ulcerative stomatitis.

Diagnosis.—If the two chief symptoms are pronounced, viz., ecchymosis and the affection of the gums, diagnosis offers no difficulty; otherwise stomacaca might be mistaken for it.

Prognosis.—The cases which have now and then spasmodically occurred in this country have always run a mild course. But the prognosis might be dubious, if bad hygienic conditions prevailed. Then the disease might take a severe course and end fatally, owing to the increasing debility or other dangerous complications (pneumonia, pericarditis, apoplexy).

Treatment.—The improvement of the hygienic conditions is essential, and the diet must be regulated. From time immemorial fresh vegetables, fruits, lime-juice, lemons, sour-kraut, etc., have been considered as the best remedies. The chief point is to obtain invigoration by suitable dietetic variety.

The local treatment is not of much avail, and is the same as in other forms of stomatitis.

(*e*) STOMATITIS MERCURIALIS (STOMATITIS FROM MERCURIAL POISONING).

Mercurial stomatitis represents the most common and practically most important kind of toxic stomatitis. Similar inflammation is sometimes observed in poisoning from lead, arsenic, and bismuth.

Etiology.—If mercury gets absorbed into the body by medical treatment (as in syphilis), or by the exercise of certain trades, or from work in laboratories and in certain manufactories; then, in certain persons, who are predisposed to it, an inflammation of the mucous membrane of the mouth occurs. The predisposition is partly due to local conditions, such as caries of teeth, uncleanliness of mouth, and partly to individual idiosyncrasy. Some people become affected by even very small quantities of mercury.

Symptoms.—It begins with all the symptoms of a catarrh, but is soon distinguished by its tendency to spread to the deeper tissues and to become ulcerated. In this respect it has much the same characters as stomacaca. Salivation is a conspicuous symptom. (See page 196.)

Diagnosis.—Having due regard to the etiology, the diagnosis is not difficult.

Prognosis.—If the affection is not very far advanced, the prognosis is good.

Treatment.—In stomatitis from medical treatment the specific cause can be discontinued, save in very severe cases. Workers in mercurial works must suspend their work, and by sweating (hot baths, etc.), the metal is in this way eliminated. Under proper local treatment the stomatitis then soon subsides. It is of great importance to have the teeth put in order and to keep the mouth well cleansed before undergoing mercurial treatment or before going to work in mercurial factories.

(f) STOMATITIS ULCEROSA TRAUMATICA (TRAUMATIC ULCER OF THE MOUTH).

Under this term is comprised a group of ulcers which owe their origin to injury, and are characterized by the ulcer being always markedly limited in extent; that is, if no secondary infection from septic matter supervenes. As etiological factors may be enumerated biting the tongue, or injury by a sharp tooth, foreign bodies, scalding and burning, etc.

In whooping-cough an ulcer may be found on the frenum, more seldom on the side of the tongue, and which is covered with fibrinous exudation, and is probably due to the rubbing of the tongue on the sharp edge of the lower teeth during the repeated attacks of coughing. In infants a similar ulcer at the point of the tongue may be seen, which is caused by the irritation from an emerging lower tooth—the so-called "dentition ulcer"; even the breaking through of a molar tooth might cause ulceration. Such small ulcers, as a rule, soon heal, as does also the "dentition ulcer"; otherwise slight painting with nitrate of silver will be found sufficient.

V. STOMATITIS GANGRENOSA (NOMA; GANGRENE OF THE MUCOUS MEMBRANE; CANCRUM ORIS).

Etiology.—It is still uncertain whether the disease is that known as an idiopathic one, *i. e.*, caused by a specific micro-organism, or whether it owes its origin to an infection by various microbes based upon a constitutional dyscrasia. It was maintained at one time that noma had a certain relation to diphtheria. That it is infectious there is little doubt, for endemics have been observed; and the disease occurs also on other parts of the body, such as the scrotum, vagina, as well as on the oral mucous membrane. The disease occurs preferably in children, and particularly in young girls of from three to twelve years of age, whose health is much debilitated through previous illnesses, or infectious diseases, measles, whooping-cough, tuberculosis, etc. Sometimes ulcerative processes in the mouth, such as stomacaca and mercurial stomatitis, are followed by noma.

Symptoms.—Noma is not often seen at its very commencement. It originates usually in the mucous membrane of the cheek, near the angle of the mouth. A hard infiltration of the size of a hazel-nut first appears, which soon softens and breaks down, forming an ulcer covered with a slimy film, and which tends to spread in its vicinity. The lymphatic glands are swollen, salivation and foetor ensue, yet the subjective comfort is not much disturbed, save for a marked apathy. Later the whole external cheek swells, becomes oedematous, shows at one or other spot a hard bluish infiltration, which also breaks down and becomes black and gangrenous. Then the cheek becomes perforated in several places, and there is practically no arresting of the terrible gangrenous process, which spreads further and further, destroying the facial skin up to the forehead, and even down the neck, leaving only stinking masses of a black color; the maxillary and nasal bones also become implicated; the teeth fall out; and, finally, after a few days, death concludes the terrible disease, with signs of general sepsis.

Diagnosis.—The diagnosis offers no difficulty after the first few hours.

Prognosis.—The prognosis is always bad. It is seldom that the gangrene spontaneously limits itself by a line of demarcation and the ulceration becomes clean. Recovery may then take place, leaving an unsightly disfigurement behind. Function may afterwards be greatly disturbed.

Treatment.—On the possibility of noma having some connection with diphtheria, an injection of antitoxin may be tried. For the rest, the mouth should be frequently disinfected, and fomentations with pure alcohol or camphorated spirit ordered. The sloughing parts must be removed. The gangrenous place itself must be very thoroughly cauterized with chloride of zinc, 20 per cent., or chromic acid, and, still better, scooped out or destroyed with the thermocautery. The keeping up of the strength of the patient presents many difficulties; it should be kept up by feeding her efficiently, most possibly by stomach-tube or by nutrient enemata, and by the exhibition of stimulants and fresh air. Sometimes, if called in early enough, we may succeed in arresting the process by cutting out the morbid part, keeping well within the healthy tissue, and then sewing the cut surfaces together (*v. Ranke*).

VI. ACUTE AND CHRONIC INFECTIONS.

1. ACUTE EXANTHEMATA.

Measles.—In measles, and also in German measles (rubeola), we see small white patches on the mucous membrane of the cheek, especially near the angle of the mouth,—first described by *Koplik*,—which are pathognomonic of the disease in its first stage, and precede the eruption on the skin.

The tongue in the acute exanthematous diseases, just as in all infections, is furred, and this depends on the peculiarity, duration, and height of the fever. In measles the white, creamy coat is thrown off during the eruptive stage in small ragged flakes, and the mucous membrane underneath then appears red and smooth.

Scarlatina.—In scarlet fever the papillae are mostly swollen and of a light red ("raspberry tongue"; "scarlet tongue").

Fever.—In high fever the tongue is covered with a brown, foetid, "*fuliginous*" coat, which has a shiny or varnished appearance (*pneumonia, typhoid*).

Variola and Varicella.—In small-pox and chicken-pox a vesicular or pustular eruption may also be seen on the palate (p. 186), the pustules showing the characteristic depression or umbilication, as elsewhere in small-pox.

2. GONORRHOEA.

Gonorrhoeal or blennorrhoeal stomatitis has occasionally been seen in new-born infants, and is caused, like gonorrhoeal rhinitis, by infection at birth from the vaginal secretions of the mother.

The mucous membrane is swollen, injected, and at some places denuded of its epithelium, or shows superficial ulceration. In the secretions and tissues gonococci may be found. Every stomatitis in new-born infants with mucopurulent secretion must awaken our suspicions as to gonorrhoeal infection.

Treatment.—This consists in cleansing the mouth by cautious swabbing out with a 10 per cent. solution of borax and touching the ulcers with lapis infernalis.

3. DIPHTHERIA.

The mucous membrane of the mouth is not often affected by diphtheria, and if it be, it is usually secondary to severe diphtheria of the tonsils and throat. It is in nowise different from the latter.

Symptoms.—There are the characteristic formations of diphtheritic membranes, foetor, salivation, and pain on eating and speaking.

Diagnosis.—The diagnosis should offer no difficulties, considering the whole clinical picture. Eventually, the finding of *Klebs-Loeffler* bacilli will settle the question.

Prognosis.—Prognosis depends on the character and severity of the attack.

Treatment.—The same as in diphtheria of the throat (see later).

4. TUBERCULOSIS AND LUPUS.

Etiology.—Tuberculosis and lupus of the mouth are, on the whole, rare affections. If at the same time tuberculosis can be found in the lung, we will then be in doubt which is the primary seat of the infection. The general opinion is that the mouth, as well as the throat, is a secondary consequence of disease in the lungs. But probably the tuberculous infection of small wounds in the region of the mouth and throat does not occur so seldom as is thought; and the tubercle bacilli are then aspirated into the lungs or bronchi and then lead to a secondary infection of these organs (*Beitzke*).

We do not deny that occasionally ordinary ulcers, for instance, those on the gums, may become tubercular in nature.

Lupus, as a rule, spreads into the mouth from the outside and occurs mostly on the lips, but sometimes also on the gums or hard palate; rarely, on the tongue.

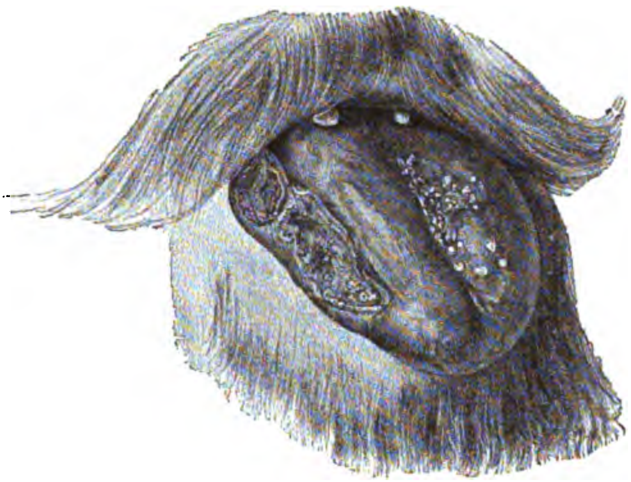

Fig. 66.—Tuberculosis of the tongue (*Hochenegg*).

Symptoms.—As in tuberculous rhinitis, the patients suffering from tuberculous or lupoid stomatitis usually visit a physician in the first place, even if ulceration has already begun. One then finds at the root (margins), or on the frenulum of the tongue or on the cheek and gums, flat, superficial or deeper, crater-shaped, sinuous ulcers, which are produced by the breaking down of miliary tubercles.

Some miliary tubercles, of the size of a millet-seed, may be seen on the edges of the ulcers. In other cases, where the tuberculosis appears as nodular infiltrations (tuberculomata), larger, sinuous, undermined ulcers of an atonic character occur. In some instances the angles of the mouth are fissured and cracked.

In lupus, small nodules of the size of a pin's head occur, which look like granulations, forming clusters, and sooner or later ulcerate. The ulcers shows nodular or condylomatous granulations on their edges and surfaces.

There is very little subjective discomfort at first. Later on, if ulceration has occurred, especially in miliary tuberculosis, pain, salivation, and foetor are very troublesome.

The regional lymphatic glands are often, though not much, swollen.

Diagnosis.—Distinction between tuberculous, gummatous, carcinomatous, and actinomycotic infiltration is not always easy, and still more difficult is the distinction between the various kinds of ulcers. *Tubercular ulcers* show an atonic character, spread more superficially than in depth, and tend to form granulations. They are seated oftener on the under surface, whereas syphilitic ulcers, on the other hand, are, as a rule, on the upper surface (dorsum of the tongue).

In lupus the ulcers are painless, granulating, and show a multitude of small nodules, which, at times, heal spontaneously with the formation of scars.

Swelling of the regional glands is much less in tubercular than it is in syphilitic or carcinomatous processes. Characteristic of *carcinoma* are the shooting pains which *soon arise* from its presence. *Never omit to examine the whole body for symptoms of tuberculosis and syphilis.* In doubtful cases, moreover, the decision will depend on a microscopic examination, which, however, as we must admit, is not always positive. A brief trial of iodid of potassium will materially help diagnosis ex juvantibus. There are also so-called mixed forms of syphilis and tuberculosis. These are influenced favourably at first by iodid of potassium, but later on the drug fails, and then the tubercular character becomes apparent.

Prognosis.—Miliary tuberculosis of the mouth often leads to a fatal ending, for it almost always exists in conjunction with the same affection of other organs; the nodular form, on the other hand, has a slow, chronic course, and not infrequently improves or heals up completely.

For the rest, prognosis must depend on the general health (condition of the lungs).

Treatment.—We must apply general and local treatment. The larger tubercles (tuberculomata) are to be removed by excision and immediate suturing; smaller infiltrations and ulcers are to be scraped out thoroughly, right into the healthy tissue, by a sharp spoon, and then cauterized with lactic acid (80 per cent. or pure). Instead of the scoop, the galvano-cautery, and instead of lactic acid, trichloracetic or chromic acid, may be used. For the pain, brushing with cocain or alypin before meals is useful. The mouth must be purified with a disinfectant mouthwash or by tablets of formamint.

5. SYPHILIS.

The mouth, as well as the throat, is a seat of predilection in syphilis, especially in the secondary stages.

(*a*) Initial sclerosis (hard chancre, primary sclerosis) often occurs on the lower lip, and more seldom on the tongue, still rarer on the gums or cheek. The infection is transmitted by contact, *e. g.*, kisses, and by infected instruments or utensils, or other accidents, difficult to explain. Sucklings are diseased by infected wet-nurses or teats. The primary hard sore soon breaks down, and forms either a superficial or deeper ulcer with hard edges, and is covered with a slimy film or crust. The regional glands are always swollen and painless; healing is slow, and the usual duration is six weeks; but the chancre can sometimes still be seen when secondary symptoms are already present.

(*b*) Secondary symptoms in the mouth are of an erythematous, papular, or ulcerative nature. The most common are the papules or broad condylomata, called "plaques muqueuses" (mucous patches). They are found as small or larger sized, round or irregular, greyish-white patches—(*a*) on the left edge of the tongue; (*b*) on the under surface, near the tip; in the mucous membrane, or somewhat raised above it, and especially on the inside of the lips, where one can see the papules of the outer skin passing into the "plaques muqueuses" of the labial mucous membrane. They occur frequently also on the mucous membrane of the cheek, at the angle of the mouth, on the tongue and lingual tonsil. Simultaneously with them the tonsils, pharynx, and soft palate are often affected. (See p. 313.)

The mucous membrane around the plaques is conspicuously red, and this is pathognomonic, but this symptom is sometimes missing. The papules quickly disappear spontaneously or under treatment; sometimes, however, they ulcerate and then leave small depressed scars behind—the so-called *scars of Erb.* They show a great tendency to relapse, often after a long interval. Under the influence of various irritations, for instance, tobacco, alcohol, hot food, etc., they incline to ulcerate. The edges of the superficial ulcers are sharply cut and indurated; the floor is irregular and is covered with a dirty, greyish-yellow film. The angles of the mouth are fissured or cracked, which should always give rise to a suspicion of syphilis if the fissures will not heal.

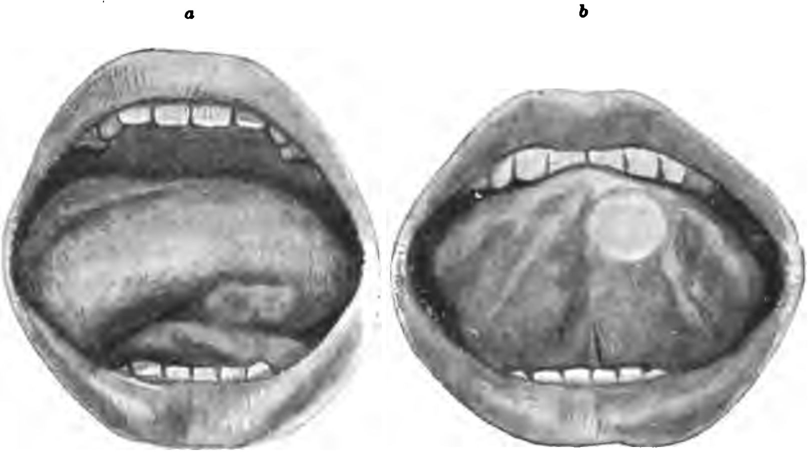

Fig. 67.—Plaques muqueuses: (*a*) On the left edge of tongue; (*b*) on the under surface.

The edges of the tongue, also, owing to the constant irritation from sharp-edged teeth, often show a fissured or corroded surface, so that the mucous patches seated on the margins of the tongue present a cracked or ragged appearance. The superficial layers of the epithelium having been (partially) cast off, the mucous membrane appears in various places, red and smooth, as though shaven, and bleeds freely when touched. After the syphilis has disappeared, often years afterwards, small, round, white patches, so-called leucomata, occur on the lips and tongue, in the region of *Erb's* scars, which must be considered as the sequelae of the previous mercurial treatment, *i. e.*, as a form of "hydrargyrosis," and are quite harmless. They should not cause anxiety on the part of the quondam patient, nor is there any reason to renew mercurial treatment, as some overcareful physicians would assert, for mercury would only aggravate the condition. Wait calmly until all the white, though somewhat obstinate, patches have disappeared, avoid irritation, and if something must be done, then paint with a weak solution of nitrate of silver or chromic acid. In simple papular syphilides the patients do not complain. On the other hand, there is much discomfort if the broad condylomata break down, causing pain and salivation. If the angle of the mouth is fissured, opening of the jaws becomes very painful.

(*c*) Tertiary syphilides are characterized by diffuse infiltration or more or less well-defined, circumscribed, tumour-like gummata, and are seated mostly on the tongue, palate, and more seldom on the lips, angle of the mouth, or cheek. They appear some long time after the infection, but may also appear quite early, especially in neglected cases, or, as I have reason to suppose, in agreement with *Hopmann* and *Chiari*, in persons run down in health and treated too long and too much with mercury. The gumma (either single or multiple) is of the size of a pea, or hazel or walnut, firm in consistency, and the surface is smooth and lies in or under the mucous membrane, which is prominent over the part concerned. The gumma breaks down and forms a flat, sharply cut ulcer, with sinuous, indurated edges; or if the process begins in the deeper tissues, the resulting ulcer is deep and crater-shaped, showing often only small fissures on its surface (fissured ulcer of *Fournier*). The ulcers, sometimes when healing, form large scars, which shrink and give the tongue a tuberous or ragged appearance (glossitis syphilitica indurativa). We must specially mention that the pharyngeal tonsil may also be the seat of tertiary syphilis, which is often overlooked.

Whether the so-called *smooth atrophy of the root of the tongue* is a symptom of late syphilis is doubtful, for it is seemingly found in old people who have never had syphilis.

On the hard palate, tertiary syphilis often affects the bone, leading to its ulceration and destruction; so that the *oral and nasal cavity are brought into communication.* In other cases the process begins in the nose (see p. 103). In small infiltrations there is not much of which to complain, but there is more, however, after ulceration has taken place. The palate being perforated, the speech becomes nasal, and food might reach the nose.

Diagnosis.—A hard chancre may be mistaken for carcinoma or gumma. In the primary affection the regional lymphatic glands are always swollen and painless, but in carcinoma the glands are also swollen, but are very tender, whereas in gumma any swelling of the glands is entirely absent. Besides the gumma softens and breaks down much earlier and more deeply. The presence of secondary eruptions will leave no doubt as to the nature of the affection.

Broad condylomata in the mouth are unmistakable, especially if in association with other signs or symptoms of syphilis (swollen glands, rashes, fissures). The differential diagnosis between syphilitic plaques, leucoplakia, and lichen planus has been discussed (see p. 191). As regards chronic hydrargyrosis, see above (p. 209).

Aphthous and herpetiform ulcers are also liable to be confounded with syphilitic ulcers, but they usually run a more acute course, are mostly painful from the first onset, and are surrounded by a more or less bright-red areola, whereas syphilitic papules and ulcers show a more subacute course, are painless, and no such areolation is seen. In herpes the vesicular eruption would hardly escape notice, and aphthae is a children's disease, seldom occurring in adults. The distinction between stomatitis mercurialis and stomatitis syphilitica secundaria sometimes offers difficulties. However, the mercurial stomatitis is always acute in its onset, and is accompanied by salivation and foetor; the peculiar affection of the gums and posterior parts of the cheek should be sufficient to insure a differential diagnosis. Occasionally both may be associated, and then the diagnosis must be secured ex juvantibus, *e. g.*, with iodide of potassium. Tertiary syphilides *sometimes* present great difficulties in regard to diagnosis. Gummata may be mistaken for tumours, cysts, deep abscesses, and, lastly, for carcinoma.

If we find the regional glands swollen, we must be on our guard and suspect carcinoma rather than benign tumours or gummata. Multiple gumma-like tumours indicate syphilis. Benign tumours are perhaps softer and more elastic than gummata, and the skin or mucous membrane covering them can be moved. For the diagnosis between primary and tertiary ulcers see p. 209, and between syphilitic and tubercular ulcers see p. 206.

Carcinomatous ulceration is considerably harder than gummatous ulceration, and its seat of predilection is the margin of the tongue; the edges of the ulcer are thickened and turned outwards. It has happened that a carcinoma has arisen on the site of a syphilitic ulcer. It may also be mentioned that cancer is a disease of older persons, whereas syphilis is a disease of younger people. In certain cases the history and the examination of the whole body will guide us; and in many others we must depend on the microscopical or bacteriological examination, and on the effect of a short trial with mercury or iodide of potassium.

Prognosis and Treatment.—With regard to prognosis and treatment, we may refer to what we have already said in Part I, in respect to the same affection of the nose. Here we shall only indicate some special points. Condylomata, erosions, and ulcers of the secondary stage are best treated by cauterizing them with chromic acid, which can be repeated as often as needful. We must warn the patient not to swallow with the saliva any of the cauterizing agent, on account of its irritant emetic action; and we should, therefore, immediately after cauterization, wash or cleanse the surface with water.

Secondary syphilis, especially the secretions of the "plaques muqueuses," is very infectious, and great attention should be paid in regard to this matter. Kissing must be forbidden; towels and other utensils used by the patient must be kept separated and for his sole use. Any irritation is to be avoided, and the mouth washed and cleaned carefully, as has already been pointed out.

In gummatous affections of the mouth, local treatment is unnecessary and worse than useless, because any cauterization which might be applied would only tend to accelerate the softening and thus also the destruction of the tissues.

6. MALLEUS, ANTHRAX, LEPRA.

Malleus and *anthrax* are seldom limited to the mouth. In malleus one finds, in combination with a similar affection of the nose, small yellowish nodules or pustules; later ulceration and swelling of the regional lymphatic glands.

Anthrax simulates the picture of gangrenous glossitis.

Lepra occurs preferably on the lips, and also on the gums and palate; and more often the pharynx shows white, nodular infiltrations of firm consistence.

Diagnosis and Treatment.—With regard to diagnosis and treatment see Part I for the same diseases of the nose.

VII. PARASITES OF THE MOUTH (MYCOSES).

Among the parasites which give rise to mycotic disease of the mouth the vegetable kingdom puts forth the most numerous agents. Occasionally also animal parasites are found, such as cysticercus cellulosae, echinococcus (hydatids), trichinae, filaria, and dracunculus medinensis (guinea-worm).

1. THRUSH.

Etiology and Pathology.—Thrush is caused by a fungus, the Oïdium albicans, which thrives best on squamous epithelium; it is, therefore, often found in the mouth of unclean infants, or in persons who are laid up with severe illness and are not kept clean, as, for instance, in pneumonia, typhoid, consumption, diabetes, etc. It is often transmitted to children during birth or in childbed, or by dirty utensils (teats). It is well known that starchy food, which bottle-fed infants are usually given (at too short intervals), on account of its liability to fermentation, forms a fertile soil for the fungi. As contagion spreads easily, epidemics, so to speak, sometimes break out among foundling children or in lying-in hospitals.

Symptoms and Course.—The disease begins with white patches like a pin's head, which unite, and soon or after a few days form greater ones of somewhat uneven surface. The patches cannot be easily separated, and are very soon renewed. The mucous membrane underneath is red, softened, and bleeds easily.

The onset is very often overlooked, as it causes no trouble; later on, pain occurs, which renders the taking of nourishment difficult. With the disease of the mouth dyspepsia or gastro-intestinal catarrh is often associated, and is probably due to the fungi being swallowed with the food. On the other hand, a preëxisting catarrh of the digestive tract might have produced a predisposition to the growth of the fungi in the mouth.

It often happens, especially if the case be neglected, that the thrush spreads into the throat, gullet, nose, and larynx, and even to the lungs or stomach. But the most severe cases are those in which the fungus, trespassing on the attachment of the epithelium, grows into the connective tissue or blood- or lymph-vessels, and is carried away into the brain or kidney, where it produces metastases.

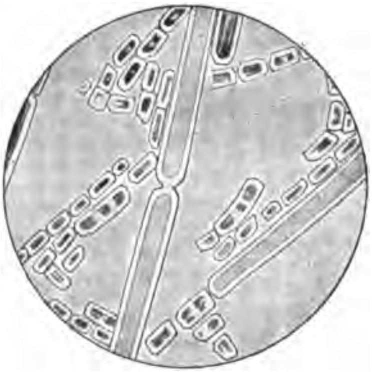

Fig. 68.—Fungus of thrush or Oïdium albicans, prepared in glycerin.

Diagnosis.—The disease, if pronounced, is easily recognizable. Should any doubt exist, a microscopical examination can be made and sets the matter beyond question.

One sees a network of many double-contoured threads, which form a chain of many links and sometimes show at their ends a club-shaped thickening. Interspersed between the network (mycelium) smaller or larger clusters of round or oval cells, of high refractile power, are seen (spores or conidia).

Confusion with remaining particles of milk, which can be easily wiped away and never form continuous patches, should not occur. Diphtheritic membranes never have such a white appearance, but look more of a greyish or brownish white, usually show a different localisation, and besides, there is fever, and the general health suffers greatly.

Prognosis is always favorable if the disease is treated in time. In weaklings, badly nourished, or in children seriously ill, the disease might become serious, and in adults also thrush is always a serious symptom.

Treatment.—Above all comes prophylaxis. In the first place, fresh air must be admitted into the sick-room, the mouth must be kept clean and well washed. (See pp. 179 and 184.) But in infants it is not good to overdo the cleansing of the mouth, so as to avoid causing *Bednar's* aphthae. Food-stuffs which are known to breed the fungi should be avoided, *e. g.*, sugary and starchy infants' foods. Complications must be treated on general lines; and if the thrush is discovered in the oesophagus, vomiting should be encouraged.

2. ACTINOMYCOSIS.

Etiology.—The etiology has been already discussed. (See Part I, p. 114.) The disease begins with small, hard, nodular swellings, which may for a long time remain quiet and painless, but finally soften and suppurate. The abscesses do not often break spontaneously; the thin pus is usually discharged after incision, and shows under the microscope, or even to the naked eye, the characteristic yellow, clubbed fungi. The process may abate, leaving simply a fistula, or retrogress, and end in scarring with retraction, while in other places the process of softening begins anew. It is mostly the lower jaw, especially the region of the salivary glands, which is affected, but the cheek also and the tongue and the tonsil may be implicated. From the lower jaw the process may spread into different regions, sometimes simulating phlegmonous inflammation.

Diagnosis.—The yellow "sulphur grains" in the pus, and the microscopical finding of a mycelial filament with clubbed ends, are unmistakable. As long as the tumour-like swellings are intact, they might be mistaken for phlegmons, gummata, or tubercular processes, and even for malignant tumours. It is advisable to search for actinomycosis in all those cases where we find a dental fistula which will not heal, or where we see on the lower jaw those chronic, insidious nodules which show no tendency to progress, and, being painless, occasionally soften and suppurate.

Prognosis.—The prognosis depends on the seat and extent of the disease. If this be localized, prognosis is not unfavourable, but it becomes bad if other important organs are affected.

Treatment.—The treatment consists in opening and thoroughly scraping out the abscesses, and giving iodide of potassium internally. Carious teeth should be removed if they do not fall out.

3. OTHER MYCOSES.

Occasionally *leptothrix* and *sarcinae* have been found in the mouth; the latter especially, under the same conditions as the thrush fungi, *i. e.*, in seriously ill and cachectic persons, but no discomfort is caused thereby.

Leptothrix prefers to attack the root of the tongue, near the entrance to the larynx (tonsillitis lingualis leptothrica); but attacks also the tonsils, the pharynx, and the teeth, in whose carious cavities they grow in enormous masses. They form on the mucous membrane white or yellowish, hard or soft points or cones, which are not easy to remove.

Sarcina grows on the mucous membrane of the tongue or throat as thin, fine, white *patches*, not unlike those produced by thrush. It has also been found in the saliva and in the stomach.

Diagnosis.—The diagnosis is not difficult with microscopical examination. Leptothrix shows a very dense mycelium, consisting of thin, fine threads or filaments lying close to each other in bundles. Sarcinae consist each of four cells, arranged like bales of cotton.

Treatment.—The treatment is the same as for thrush. (See also Pharyngomycoses, p. 317.)

VIII. TUMOURS.

Etiology.—We find tumours on the lips and alveolar margin at the gums; most frequently on the tongue, *i. e.*, on those parts which are most exposed to injury and irritation.

What rôle acute or chronic inflammation, in the widest sense of the term, plays in the etiology of tumours is not quite ascertained. *Von Hansemann* suggests that malignant tumours are generated on a predisposed site by external irritation. Other investigators consider that cancer, which occurs so often on the lips and tongue, is due to parasites. In any case this fact is certain: that cancer of the lips and tongue is very common with smokers, and that on the site of quite innocent erosions ulcers and tumours develop which are frequently exposed to irritation through the sharp edges of teeth, hot food, caustics, etc. Syphilis and leucoplakia also are said to predispose to the development of carcinoma. Cystic new-growths are usually retention tumours caused by the blocking of the salivary ducts.

1. TUMOURS OF THE LIP.

Besides the rare *muco-cysts* on the inner side of the lip and the *fibromata*, as also certain congenital or acquired hypertrophies, we have mostly to deal here with *angioma* and *carcinoma*. The former is usually seated at the angle, the latter, more towards the centre of the lower lip.

Cancer of the lower lip begins as a small node, which, however, soon softens and forms an indurated ulcer with a fissured or cracked surface. The regional glands swell early.

Diagnosis.—Confusion is possible with—(*a*) hard chancre (which never shows such a cracked or fissured surface); (*b*) with secondary syphilitic ulcer (less hard, seated on the angle of the mouth, other secondary syphilitic signs present); (*c*) *eczematous fissures*, showing sometimes hard edges, but *no swelling of the glands.*

By pressure on the carcinoma one is often able to press out the so-called cancerous milk, viz., small white cylindric or conical masses of epithelial cells, as in comedones. In any case microscopical examination of excised portions of the tumour will reveal its nature.

Treatment consists in early extirpation and afterwards plastic operation. In recent times x-rays and radium treatment have been successful in those cases where the cancer was not very advanced and not complicated with glandular swellings. The thermo-cautery and 50 per cent. chromic acid (*Blaschko*), as also arsenic in solution internally, have been used.

2. TUMOURS OF THE GUMS.

Tumours of the gum which spring from the periosteum of the alveolar process are grouped somewhat summarily under the collective name of epulis. Their structure is that of a fibroma, sarcoma, or carcinoma. They can be seen in the mouth as smaller or larger tumours, stalked like a mushroom or sessile. The tumours grow around the teeth, which are at length loosened and fall out. Occasionally, dental cysts occur; and quite seldom odontomata which develop before the second dentition has finished. (See p. 162.)

Epulis may be confounded with the parulis, commonly called a "gumboil." The latter is a subgingival, subperiosteal abscess, due to periostitis in dental caries, and can be felt as a tender, at first tense, later on fluctuating, swelling outside the alveolar margin. It is nearly always accompanied by oedema of the cheek, and in extreme cases, oedema of the eyelids.

3. TUMOURS OF THE TONGUE.

Lympho-cavernoma (macroglossia) has been already mentioned (p. 194). There occur also angioma, lipoma, fibroma, etc., and mixed tumours. They do not cause much discomfort; but this depends mainly on the size of the tumour.

Treatment.—The treatment consists, according to size and seat, of removal of the tumour by scissors, knife, or snare.

The *cysts of the tongue* and *hypertrophy of the lingual tonsil*, though the latter is not really a tumour, sensu strictiori, will now be discussed separately.

(*a*) **Cysts.**—Underneath the tongue, on the floor of the mouth, and usually on one side of the froenum, a grey-white, translucent, and fluctuating tumour is sometimes found, which is termed a *ranula* (little frog's tumour), and the pathology of which is still in question. We have probably, in most cases, to deal with a congenital formation or with a retention cyst of the salivary glands.

The tongue is pushed upwards, so that speaking and chewing or even respiration may be impeded. Other cystic formations are sebaceous and dermoid cysts, hydatids, (echinococcus cysts).

Treatment.—Consists of excision of the whole cyst. This will be successful if the cyst-wall is thick enough; otherwise, as in ranula, the cyst is incised and the contents are let out, and then a portion of the cyst-wall is excised. The margins of the cyst are then stitched to the cut edges of the wound of the mucous membrane. The sutures are removed after four or five days. Instead of sewing as above, one may plug the cyst daily with sterile gauze.

(*b*) **Hypertrophy of the Lingual Tonsil.**—*Etiology.*—Hypertrophy of the lingual tonsil is due to frequently repeated inflammations or chronic irritation in the posterior region of the mouth. Most commonly the hypertrophy of the lingual tonsil is only a part of the chronic inflammation, which is also seen in other sections of the pharyngeal lymphatic tissue. We find, therefore, swelling of the lingual tonsil in scrofulous struma of children, in women at the age of puberty, or at the climacteric. Apparently, the lingual tonsil is oftener enlarged in adults than in children, in opposition to the faucial tonsils, which are most commonly enlarged in childhood. This difference can, perhaps, be explained by the circumstance that the hypertrophied lingual tonsil in children is less often sought for than it is in later years, owing to the lack of nervous or other complications.

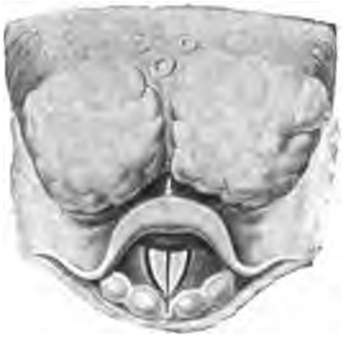

Fig. 69.—Hypertrophy of the lingual tonsil.

Symptoms.—The patients complain usually of a sensation of tickling or fulness in the neck, of irritation in the throat which makes them cough, of retching, or of "hawking and spitting," and of becoming tired when speaking. In women, all sorts of nervous troubles occur (globus hystericus, etc.). In other cases all symptoms are wanting, and the hypertrophy is discovered only by chance. (See Fig. 69.)

On examination, having the tongue sufficiently depressed. one sees either directly, or, still better, indirectly by the throat mirror, single or several globular or pear-shaped swellings close to each other, which, in extreme cases, fill both valleculae and may compress the epiglottis, causing difficulty in using the voice, especially in singing. Ectatic and varicose veins are often seen on the swollen parts, and these veins occasionally burst and thus give rise to haemorrhage.

Diagnosis.—The diagnosis is easy, as one is able to palpate with the finger-tip the hardish prominences. They are not likely to be mistaken for real new-growths or aberrant lobules of thyroid gland, both of which are occasionally found.

Prognosis.—The prognosis is favorable, for the lingual tonsil is freely accessible.

Treatment.—An hypertrophy, discovered by chance, should be left alone. Should there be symptoms of trouble, operative measures are far and away the best, as painting or brushing are very annoying and mostly useless. Possible varicose or ectatic veins must be avoided. According to the extent of the enlargement, scissors, double curette, tonsillotome, or the snare may be used. In some cases the hypertrophy can only be removed piecemeal or during several sittings.

MALIGNANT TUMOURS.

Sarcoma of the tongue seldom occurs, but cancer is comparatively frequent.

CANCER OF THE TONGUE (CARCINOMA LINGUAE).

Etiology has already been discussed.

Symptoms and Course.—It is not often that the carcinoma at the outset arises as a tumour in the substance of the tongue, and then grows towards the surface, where it is prominent, and finally softens and ulcerates, the ulcer being indurated, its edges thickened, hard, and pad-like; but much more commonly it arises originally from a fissure, scar, or wound, or from a papilloma as a result of carcinomatous degeneration. On the one hand, the cancer starts, ab initio, as an ulcer; on the other hand, the ulceration is the result of breaking down of a distinct tumour.

The malignancy of the new-growth shows itself by the increasing induration of its base and by the swelling of the regional (cervical) glands, which latter, however, may not occur until comparatively late in the disease. The ulceration unceasingly spreads and destroys the tongue, as well as invading adjacent organs. There is also considerable pain at night, which radiates into the ear, and foetor ex ore appears. Speaking and feeding are rendered difficult, and salivation and occasionally also haemorrhages, occur. Increasing cachexia or a pneumonia finally puts an end to the misery in the course of one year to eighteen months.

Diagnosis.—If the clinical symptoms are doubtful, microscopical examination will decide the question. (See p. 210.)

Prognosis.—The prognosis is bad, for even after early operation, recurrences are very frequent.

Treatment.—We must endeavour, as early as possible, to radically remove all the morbid growth right down into the healthy tissues, if necessary, by amputation of the tongue and extirpation of all visible lymphatic glands. In inoperable cases, besides the usual symptomatic treatment, *x*-rays or radium treatment can be administered.

Prophylactic inspection of the tongue in regard to possible or existing ulcers or losses of substance or other pathological appearances is desirable.

Every wound or wart which does not heal after two or three weeks after the causative factors (smoking, alcohol, sharp-edged teeth, etc.) have been removed, particularly in older people, should be radically excised or destroyed by the thermo-cautery. Too frequent painting with caustics should be avoided, because it is likely to determine the outcome of a carcinoma.

IX. NERVOUS DISORDERS.

1. DISORDERS OF TASTE.

(*a*) **Ageusia and Hypogeusia.**—Complete abolition (ageusia) and diminution (hypogeusia) of taste are due to either peripheral or central lesions. Peripheral causes are: *Diseases of the* *oral mucous membrane, and, consequently, that of the terminal organs of taste; diseases of the nose,* which diminish through nasal obstruction, the so-called gustatory smelling (see p. 14), and, owing to dryness of the mucous membrane of the mouth, render gustatory stimulation difficult; *diseases of the middle ear,* which affect the chorda tympani and the plexus tympanicus; *dyspepsia* (through furred tongue); *lesions of the gustatory cups* directly through too hot or cold food or beverages, drugs (cocaine, gymnema, etc.).

Central causes are: *Intracranial diseases* in the region of the fifth nerve, *hysteria,* and *traumatic neurosis.*

Disturbance of taste varies according to the seat of the lesion, and is unilateral or bilateral, and concerns the anterior part and margin of the tongue if the lingual nerve and the chorda tympani are affected; or it occurs in the posterior part (root) if the glosso-pharyngeal nerve is diseased. The sensation of the various shades of taste may be affected in unequal degrees.

(*b*) **Hypergeusia.**—An exaggerated gustatory sense is often only an increase of the normal sense of taste, but occurs pathologically also, especially in hysterical persons.

(*c*) **Parageusia.**—Here certain shades of taste are distinguished in a faulty manner, and mostly unpleasantly. It is mostly a certain quality, which always recurs. All things taste bitter, salty, or foul. It is often met with in hysteria and pregnancy. In other cases this is due to febrile and gastric disorders in which the tongue is heavily furred. Lastly, there are certain mental diseases which are accompanied by hallucinations in the sphere of taste, or the latter form only the stage of irritation preceding the onset of paralysis (ageusia).

Diagnosis.—For examination of taste, in the way already described, with regard to the sense of smell (see p. 16). A more accurate diagnosis in respect of the cause and seat of the lesion can only be made after longer observation.

Prognosis.—The prognosis depends on the cause and seat of the lesion.

Treatment.—Must take into account the cause, and local treatment, if at all successful, could only consist in the administration of the electric current.

2. LESIONS OF SENSIBILITY.

(*a*) **Anaesthesia.**—Complete abolition or diminution of sensibility occurs almost only in hysteria and in certain diseases of the brain or in peripheral paralyses of the second and third divisions of the trigeminal nerve, and are usually associated with disorders of smell. Hysterical and peripheral anaesthesiae are mostly unilateral, and central paralyses often bilateral. The insensitive tongue is easily caught between the teeth and bitten, thus showing lesions; particles of the food remain, without attracting notice, on the tongue, between the cheek and teeth, giving rise to decomposition; and even differences of temperature are not perceptible to the patient.

(*b*) **Hyperaesthesia,** which is often very acute in the tongue, and may even reach the degree of neuralgia, known as glossodynia or glossalgia, is less frequently due to hysteria or central disease, but is more often caused by neurosis of the fifth nerve; in some cases it takes the form of radiating pains, the cause of which can be traced to affections of the throat, *i. e.*, lateral pharyngitis or in enlargement of the foliated papillae. Glossodynia has also been observed in gastric or rheumatic troubles and in cases of chlorosis and anaemia.

Glossodynia occurs in paroxysms, spontaneously, or on the attempt to speak; or it is continuous and sometimes disappears by itself.

(*c*) **Paraesthesia** is characterized by various unpleasant sensations in the tongue, *e. g.*, numbness, heat, cold, tingling, itching, pins and needles, etc., and is usually associated with other nervous troubles.

Treatment.—Is unavailing if the cause is inaccessible. (See also p. 125.)

3. DISORDERS OF MOTILITY.

(*a*) **Paralysis.**—Paralysis is either complete or there is only a defect in motility (paresis).

Etiology.—Paralysis of one or several muscular groups is often of central origin, viz., due to disease of the brain, as in apoplexy, tumour, syphilis, etc., or of the medulla oblongata (bulbar paralysis, locomotor ataxia, and diphtheria) (?). Peripheral paralyses in the region of the mouth, with the exception of the lips, seldom occur.

Symptoms.—Paralysis of the labial muscles, usually unilateral, renders speaking difficult, and blowing up the cheeks impossible. The angle of the mouth droops, and the saliva runs out.

In paralysis of the muscles of mastication the patient has difficulty in chewing, and in bilateral paralysis the lower jaw drops.

Paralysis of lingual muscles, often the first sign of bulbar paralysis (paralysis glosso-labio-pharyngea), inhibits articulation, chewing, and swallowing; the tongue itself lies flabby and flat on the floor of the mouth and is thickly furred. Because the tongue can not be elevated, food often slides forwards from the pharynx in swallowing. Paralysis of longer duration leads infallibly to atrophy.

In paresis, which often precedes paralysis, the symptoms are less marked. In unilateral paralysis the tongue is pushed over towards the paralysed side, and small fibrillary twitching of the muscles may often be noticed.

Prognosis.—The prognosis is in any case doubtful, and the more so, the greater the difficulties we experience in removing the cause, and the longer and more complete the paralysis. *Hysterical* and syphilitic paralyses have better chances of recovery; the same may be said of those due to apoplexy, which sometimes disappear spontaneously. Peripheral paralyses usually allow of a more favourable prognosis.

Treatment.—Must take into consideration the causes, and locally the electric current may be applied.

(*b*) **Spasms.**—Clonic spasms (of short duration and interrupted by short intervals) in conjunction with tonic (continuous) spasms, occur in the lips and tongue in various nervous diseases (*e. g.*, epilepsy, chorea, hysteria, etc.). Idiopathic spasms of the tongue alone, on the other hand, are very rare. In such cases the tongue wags, is painful, and pressed towards the palate, and speaking and swallowing are rendered difficult or impossible. Tonic spasms of the masticatory muscles (trismus, lockjaw) occur in tetanus, epilepsy, or meningitis, and both jaws are clenched firmly together. The clonic form (masticatory facial spasm) is characterised by rhythmic movements of the lower jaw, usually in vertical direction (chattering), less often horizontally (grinding).

Prognosis.—Depends on the etiology.

Treatment.—Quite apart from the causal treatment, must aim at removing the excitory irritation (carious teeth). In tetanus injections of antitoxin, administration of narcotics, and artificial feeding. In other cases the electric current might be usefully applied.

X. DISEASES OF THE SALIVARY GLANDS.

I. INFLAMMATIONS.

The salivary glands, either singly or several together, may be the subjects of primary disease; as, for instance, in epidemic parotitis; or the submaxillary gland may be affected in a similar manner. They are also liable to secondary disturbances, as part of a general disease, or to blocking or inflammation of the ducts. The blocking of their ducts only leads in some cases to a transient swelling of the glands, and this "tumour salivalis," as it is called by *Schech*, subsides if the duct again becomes permeable. In one case, which I myself observed, the swelling of the parotid gland was periodical, and followed on the use of potassium iodide. In other cases a chronic enlargement occurred, especially of the submaxillary and sublingual glands. I had the opportunity to observe a case of chronic parotitis hypertrophica in a woman who was repeatedly attacked by mumps as a result of carious teeth. The disease first described by *Mikulicz* and called after him, which consists in a symmetrical swelling of all the salivary and lachrymal glands, is likewise a chronic affection of the glands, and is probably only a symptom of the so-called pseudoleukaemia, or *Hodgkin's* disease. This harmonises with the fact that in not a few cases arsenic and the x-rays had a decidedly good effect.

(a) Inflammation of the Parotid Gland (Parotitis; Cynanche Parotidea).

Etiology and Pathology.—Inflammation of the parotid is either primary—idiopathic; or secondary—metastatic. The first is usually an epidemic infectious disease, called epidemic parotitis ("mumps"), which occurs mostly in children or young adults, and never suppurates. The secondary, or metastatic, inflammation occurs as a complication of serious infectious diseases, *e. g.*, typhoid, scarlet fever, pneumonia, pyaemia, tuberculosis, and also in carcinoma, and nearly always leads to the formation of an abscess. In both groups the germs of infection probably invade the gland through the *Stenonian* duct from the mouth. The incubation in the epidemic form occupies from three to twenty days.

Symptoms and Course.—The disease begins often, but not always, with slight prodromal fever, a feeling of tension and pain below and in front of the ear. A swelling soon appears below and in front of the ear, which reaches behind to the mastoid process, below to the angle of the jaw, thus broadening and disfiguring the face. The other glands are sometimes involved, especially the submaxillary and sublingual glands. There are cases in which the disease is localised even to the submaxillary, and it may happen that in one family one member gets a swelling of the parotid and the other only of the submaxillary gland.

The subjective symptoms are a feeling of tension and pain, especially on the attempt to open the mouth, radiating into the neighbourhood, and the neck is stiff, or the head may even be drawn to one side. There is sometimes salivation or stomatitis, owing to the mouth being inefficiently cleansed because of the pain.

The course is always short; fever is moderate, and increases only if the other glands are affected or other organs are implicated. The most common complication is an inflammation of the testicles (orchitis); usually one testicle only, and mostly the right, is affected; sometimes also the epididymis. The orchitis, though often very painful, never passes on to suppuration, but sometimes atrophy, and consequent impotence, may follow the inflammation.

In women an analogous swelling, though not so frequent, is found in the breasts, and of the internal and external genital organs, especially of the ovaries. We still lack the explanation of the connection between the parotid and genital glands. Other complications are occasionally observed as the result of pressure on the neighbouring organs or vessels, *e. g.*, buzzing noises in the ear, difficulty of hearing and in swallowing, hoarseness, dyspnoea, etc. Further complications again, *e. g.*, endocarditis and pericarditis, arthritis, meningitis, are probably due to the same infection. Such severe complications, however, very seldom occur, and the average course of the disease takes from eight to fourteen days, but often not so long.

Metastatic parotitis takes, *from the onset*, a much more serious course. It usually leads to suppuration, and involves the facial nerve. The abscess, if not artificially emptied, bursts outwardly or into the ear, or the suppuration affects other neighbouring organs, leading to serious complications.

Diagnosis.—The diagnosis, with due respect to what we have said, is easy.

Treatment.—Epidemic parotitis does not require special treatment, apart from complications. In order to relieve the tension, an ointment of olive oil or other embrocation can be rubbed on. Abscesses should be early incised, care being taken to avoid the facial nerve and blood-vessels. Isolation of the patient may be desirable.

(b) **Inflammation of the Submaxillary and Sublingual Glands (Lymphadenitis Submaxillaris et Sublingualis).**—The inflammation of the submaxillary gland, concomitant with or replacing epidemic parotitis, has been described in the previous chapter. (See p. 224.)

We see submaxillary lymphadenitis frequently occurring in the course of acute infectious diseases, as in scarlet fever, measles, angina, diphtheria, stomatitis, etc., especially in so-called mixed infections, and it leads comparatively often to suppuration. The sublingual gland is less frequently affected.

Treatment.—The treatment at the commencement must consist of the application of hot fomentations, especially in the sublingual inflammation, also in the use of warm gargles. I have in many cases observed the subsidence of the glandular swelling after the use of Bier's treatment. Abscesses must be opened early.

(c) **Pfeiffer's Glandular Fever.**—*Etiology and Pathology.*—"Glandular fever" is an infectious disease peculiar to children, which is characterised by an inflammatory swelling of the superficial and the deep lymphatic glands of the back and front of the neck. The pathogenic germ is not yet known. It is probably due to streptococci entering the body by the tonsil, or more probably through the adenoid tissue of the nasopharyngeal space; perhaps the influenza bacillus plays a part. This would at least explain the epidemic occurrence of the disease. *Hochsinger and Zappert* consider glandular fever as the result of an inflammation of the pharyngeal tonsil.

Symptoms.—The disease commences with high fever, vomiting, and shooting pains, and sometimes indefinite pain in the neck. The neck is stiff, or the head is turned to one or other side. If we examine the throat, nothing abnormal except redness can be found; but the jugular and cervical glands, especially those which lie in front of or behind the sterno-cleido-mastoid muscle, are swollen and tender. The submaxillary glands may also be involved, but the swelling of the lateral and posterior cervical glands is characteristic, and this accounts for the wry-neck.

Prognosis.—The course is always favourable. After a few days the fever abates, the swelling of the glands subsides, though relapses are not infrequent. It often happens that one or other swelling persists for a long time and simulates scrofulosis.

Treatment.—According to *Heubner*, the course of the disease can be shortened by a "sweating cure," and the administration of quinine per orem or rectum for two or three days continuously. I prefer aspirin, ½ to 1 tablet per diem (5-grain tablet).

(d) **Angina Ludovici (Ludwig's Angina; Cellulitis of the Floor of the Mouth).**—*Etiology and Pathology.*—This disease, first described by *Ludwig*, a Würtemburg physician, in 1836, and called after him, is a phlegmonous inflammation of the floor of the mouth, caused by pyogenic organisms which enter the body by a wound of the oral mucous membrane or by carious teeth. The submaxillary gland is often implicated.

Symptoms and Course.—The disease commences with a swelling in the region of the submaxillary gland, accompanied, or oftener unaccompanied, by fever. The swelling quickly spreads over the whole floor of the mouth and neighbouring part of the neck, and is very tense; opening of the mouth and swallowing become difficult, and respiration may be impeded by compression or oedema of the entrance to the larynx. The skin over the swelling reddens, softens, and finally gives way, and pus is discharged spontaneously or artificially outwards or into the mouth (cynanche gangrenosa). In rare cases the inflammation subsides without the formation of pus.

Diagnosis.—The diagnosis is easy; the physician must *not, however,* confound the disease with isolated phlegmon of the submaxillary gland or actinomycosis. In the former the swelling is confined to the gland, and in the latter a microscopical examination will prove decisive.

Prognosis.—The earlier we can do something, the more favourable it will be. In very acute, but, fortunately, not frequent, cases, death has occurred with signs of pyaemia or septicaemia, and in other cases cold abscesses have followed in the neck.

Treatment.—At the commencement we may try to check the process by the application of ice, but this is usually of little avail, and then we have soon to resort to softening methods by warm applications *and an early incision.* In cases of oedema of the larynx threatening suffocation tracheotomy might be required.

II. SALIVARY CALCULI.

Etiology and Pathology.—Salivary calculi, of the size of millet seeds to that of a walnut, are hard masses, consisting of lime salts, and are usually found in the ducts of the submaxillary or sublingual, and more seldom of the parotid, gland. Usually small foreign bodies, which have gained access from outside or from the mouth, give rise to the formation of calculi, or the exciting cause is a fungus (leptothrix buccalis), especially if pathological changes in the glands or their ducts foster the colonisation of the fungi.

Symptoms.—Obstruction of the salivary ducts leads to painful swelling of the glands, eating is rendered difficult, particularly in cases of calculi in Wharton's duct. If the calculus is not discharged spontaneously or removed artificially, suppuration, with the formation of a fistula, may ensue.

Diagnosis.—The diagnosis is based on the finding of hard, tuberous masses, either by palpation or by probing. If *Wharton's* duct is blocked by a calculus, the tongue is pushed up, and the pain occurs paroxysmally. Calculi in the sublingual duct are sometimes visible underneath the tongue, as white, hard, shiny bodies, which rarely cause pain. They are not easily mistaken for tumours.

Treatment.—Expression of the concretions by massage or direct pressure rarely succeeds, and it is mostly necessary to make an incision. Sometimes the incision of an abscess caused by a calculus will also serve to remove the calculus.

III. TUMOURS.

Benign tumours occurring in the parotid gland are cysts and chondromata; *malignant growths* are sarcoma and carcinoma. The other salivary glands are rarely the seats of new-growths.

Symptoms.—Tumours of the parotid disfigure the face and render the taking of food painful and difficult, or involve adjacent organs (facial paralysis, deafness, etc.).

Diagnosis.—The diagnosis is not always easy. On the whole, tumours of the parotid simulate, to a certain degree, the hypertrophic enlargement of the gland.

Prognosis.—The prognosis corresponds to the size, and nature, and duration of the new-growth.

Treatment.—Must probably always be surgical. With regard to the chances of actinotherapy, not much can at present be decided.

PART III.

Diseases of the Pharynx.

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Diseases of the Pharynx.

GENERAL SECTION.

I. ANATOMY.

The throat (pharynx) forms a tube, which lies in front of the vertebral column, and is attached to it by loose connective tissue (fascia praevertebralis), and narrows towards its lower end like a funnel. It reaches from the base of the skull to the oesophagus, and communicates in front with the nose, Eustachian tubes, mouth, and larynx. One accordingly distinguishes three sections, the upper or nasal part (naso-pharyngeal space, cavum pharyngonasale, epipharynx), the middle or oral part (pars oralis, mesopharynx), and a lower, so-called laryngeal part (pars laryngea, hypopharynx).

(*a*) PARS NASALIS (NASO-PHARYNGEAL SPACE; EPIPHARYNX).

The nasal part of the pharynx forms a space varying in size—in adults it is of about the capacity of a walnut. Its upper wall, the roof of the pharynx (fornix pharyngis), is firmly attached to the fibro-cartilago basilaris (basal cartilage), and, forming an arch, is continuous with the posterior wall, which corresponds to the atlas and axis. The anterior arch of the atlas often projects into the lumen of the naso-pharyngeal space, which circumstance might sometimes mislead when making a posterior rhinoscopy or in digital examination.

It is in communication in front with the nasal cavity through the choanae; below these is the soft palate (velum palatinum), which, if contracted, forms the lower wall of the epipharynx. If the soft palate is flaccidly hanging down, the nasal part of the pharynx is continuous with, or passes directly into, the mesopharynx.

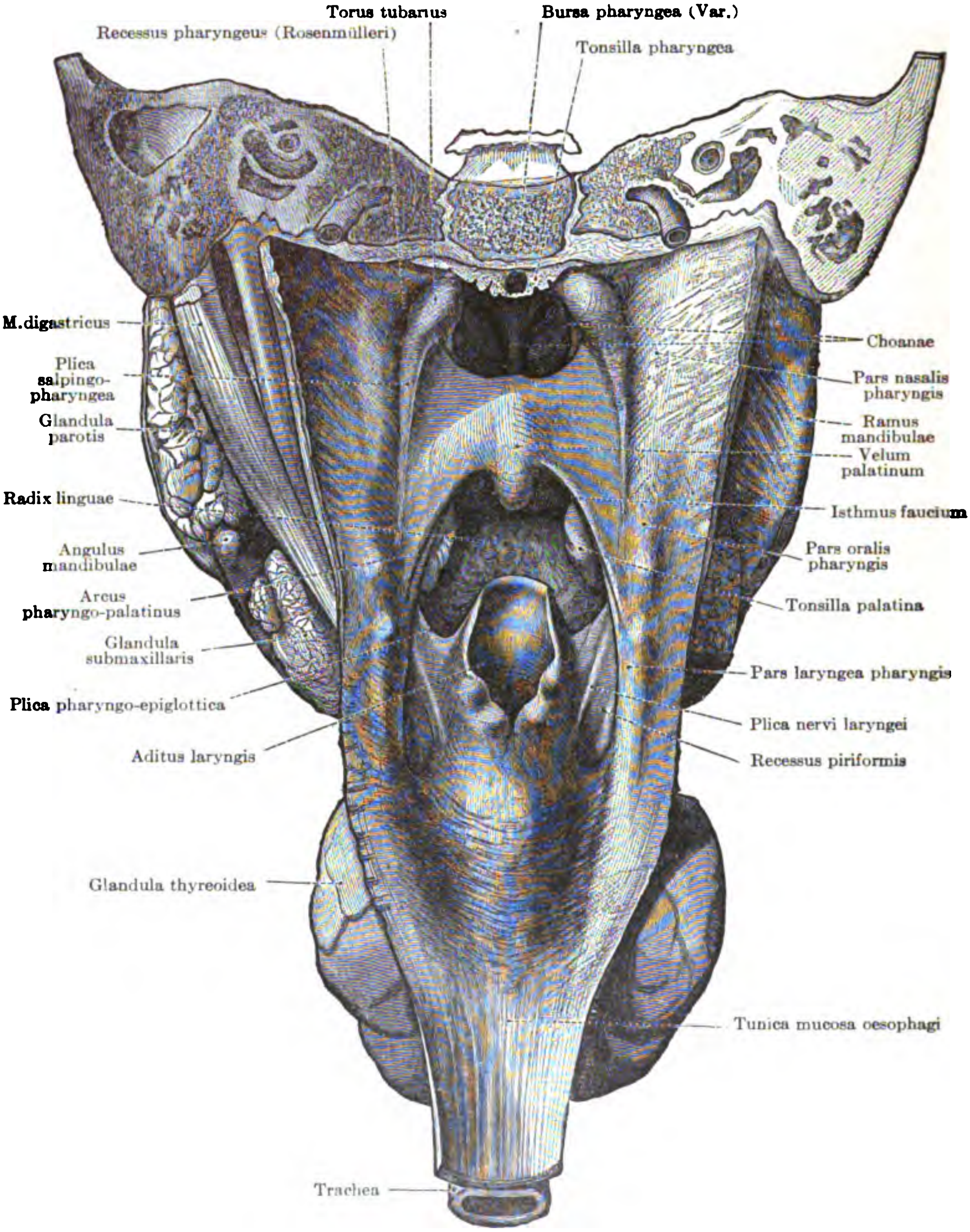

Fig. 70.—Pharynx viewed from behind (after *Toldt*). The posterior wall of the pharynx is divided in the middle line; and both parts are turned back.

The Eustachian tube (tuba Eustachii) opens into the lateral wall by a small triangular or chink-like opening (ostium pharyngeum tubae), which lies surrounded by a padded prominence, the torus tubarius, ½ cm. behind the attachment of the lower turbinal bone. The torus tubarius, which contains the tubar cartilage (cartilago tubaria), projects somewhat into the pharynx; and behind it a groove or depression may be seen, called the pharyngeal recess, or *Rosenmüller's* recess (recessus pharyngeus seu *Rosenmülleri*).

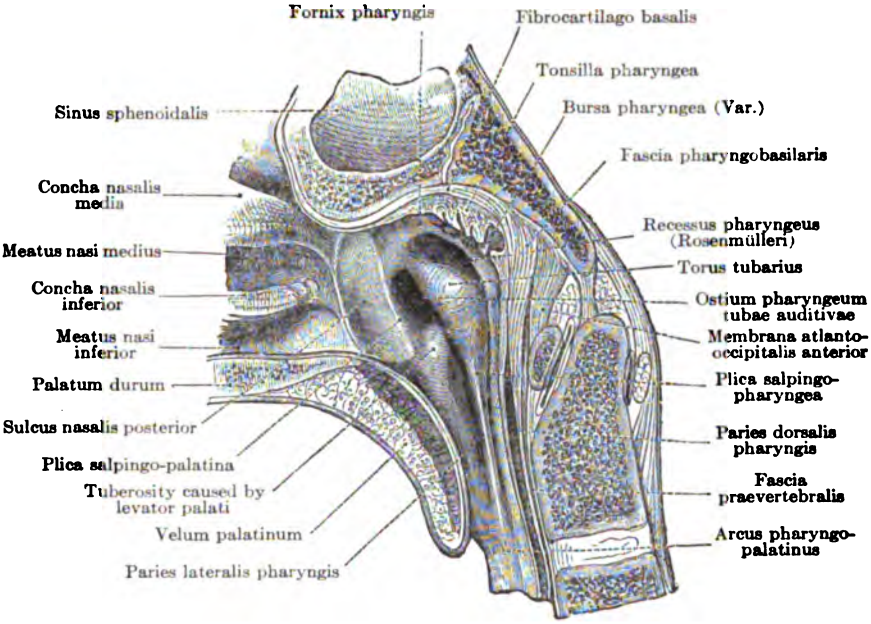

Fig. 71.—The naso-pharyngeal space (after *Toldt*) (right lateral view).

From the posterior, thicker, and more prominent part of the torus (labium posterius) the plica salpingo-pharyngea (tubo-pharyngeal fold) passes downwards, the lower part of which can be seen as a red stripe on the lateral wall of the pharynx under pathological conditions. From the anterior and smaller branch of the torus (labium anterius seu hook) descends the plica salpingo-palatina or hook-fold, ending on the back (dorsal) surface of the soft palate. (See Fig. 70.)

The m. levator veli palatini projects like a pad from below into the niche formed by the two folds (eminentia m. levatoris). (See Fig. 71.)

The mucous membrane of the naso-pharyngeal space, in the region of the fornix, is covered with ciliated epithelium, and shows an adenoid, *i. e.*, honeycombed arrangement, containing many lymph-crypts composed of leukocytes and lymphocytes or follicles. It forms there a soft cushion, divided or split up sagittally into numerous processes or projections, which, in particular cases, unite superficially and thus form a pocket or bursa (bursa pharyngea). This split-up portion of the mucous membrane is termed *pharyngeal or third tonsil* (tonsilla pharyngea or *Luschka's* tonsil).

The pharyngeal tonsil is mostly found in children; at the age of puberty it begins to atrophy and is rarely met with after thirty. In adults several recesses may be seen, representing the vestiges of the pharyngeal tonsil.

(*b*) PARS ORALIS PHARYNGIS (MESOPHARYNX).

The oral part of the pharynx reaches downwards to a plane taken through the hyoid bone, and communicates with the mouth in front by the isthmus faucium. The isthmus is formed above by the soft palate with the uvula; below, by the root of the tongue with the lingual tonsil; and laterally, diverging from the uvula, by the palatine arches. One distinguishes the two posterior arches (arcus pharyngo-palatini), which pass towards the vertebral column into the lateral walls of the pharynx, and the two anterior arches (arcus glosso-palatini) which pass on to the edges of the tongue somewhat higher and more laterally than the first two. Between both arches at each side lies the tonsil (tonsilla palatina), in the sinus tonsillaris, which is deeper above the tonsils and forms there the fossa supratonsillaris.

The soft palate, the direct continuation of the hard palate, is formed by a duplicature of the mucous membrane, which contains between its layers bundles of muscles, and which also sends a process of varying length downwards—the uvula. The anterior surface of soft palate is covered by squamous epithelium, carrying some taste-buds. The mucous membrane contains many mucous glands, and with their mouths showing small elevations, frequently covered with a drop of mucus. On the upper (dorsal or nasal) surface the mucous membrane of the velum shows ciliated epithelium, which is continuous with the squamous epithelium of the uvula.

The (palatine) tonsils are formed by conglomerations of adenoid tissue and are a part of the lymphatic ring of the pharynx. They vary very much in size and shape. Their surface shows, like a sponge, numerous round or slit-like openings, leading into equally numerous crypts or lacunae. The occurrence in them of cartilaginous or osseous fragments can frequently be noticed; and muscular fibres also have been found in the tonsils.

According to *Stöhr*, under physiological conditions, there is a constant migration of leukocytes or lymphocytes through the lacunae or spaces of the epithelium; and by the same way bacteria may penetrate into the tissue. Thus the (palatine) tonsils form the main gate for the entrance of pathogenic germs (streptococci, diphtheria and tubercle bacilli).

Waldeyer's lymphatic ring, which encircles the isthmus faucium, is essentially composed, as we have seen, of the pharyngeal, palatine, and lingual tonsils; and in these, the lymphatic tissue of the throat is mostly accumulated. But we can also find lymphatic tissue further afield from the pharyngeal tonsil, particularly in *Rosenmüller's* fossa, and also in the posterior pharyngeal wall, and not infrequently, some coxcomb-shaped nodes of adenoid tissue pass from the palatinal gland on to the root of the tongue, right to the lingual tonsil, thus actually closing the lymphatic ring. This, however, occurs mostly in those cases in which the adenoid tissue is hyperplastic, and exceeds the physiological limits. The lymphatic granulations, frequently found on the posterior pharynx, nearly always indicate a hyperplastic pharyngeal tonsil.

(c) PARS LARYNGEA PHARYNGIS (LARYNGEAL PART OF THE PHARYNX; HYPOPHARYNX).

The laryngeal part of the pharynx passes into the oesophagus at a plane which is taken through the lower margin of the cricoid cartilage. The anterior wall of the hypopharynx is partially formed by the posterior wall of the larynx, which communicates with the throat by the aditus laryngis, covered by the epiglottis. At each side of the aditus lies a pocket formed by the mucous membrane, called sinus or recessus pyriformis, which leads into the oesophagus; a fold can be seen in each sinus, called plica nervi laryngei superioris (fold of the superior laryngeal nerve). The pharyngo-epiglottic ligaments (ligamentapharyngo-epiglottica) extend from the epiglottis to the lateral pharyngeal wall, forming the border-line between the valleculae and the pyriform sinus.

Muscles of the Pharynx.—1. *Constrictores Pharyngis.*—There are three constrictors: MM. constrictores pharyngis superior, medius, et inferior. Their action is to constrict or narrow the pharynx, and thus to effect the gliding down of the morsel of food. The upper fibers of the superior constrictor have a special function. They form, by contraction, a curved projection on the posterior wall of the pharynx, the so-called pad of *Passavant,* against which the soft palate is lifted by the contraction of the m. levator veli palatini, and pressed; and so, by the joint action of these two muscles (levator palatini and superior constrictor pharyngis), the naso-pharyngeal space is effectually shut off from the mouth (pars oralis pharyngis).

2. *M. Stylopharyngeus.*—It is attached to the styloid process above and is inserted below—by one (superior) portion, into the lateral wall of the pharynx; and by the second (inferior) portion, onto the epiglottis and upper margin of the thyroid cartilage. The muscle enters the pharynx between the superior and middle constrictor of the pharynx.

3. *M. Veli Palatini.*—The soft palate is moved by five muscles: two of them, the levator and tensor, arise from the base of the skull. The levator arises from the apex of petrous pyramid and the tensor from the sphenoidal bone. The action of the latter (tensor) is to render tense the velum, by shortening its tendon, which is reflected round the hamular process; the former (levator) acts as an elevator. By their attachment to the Eustachian tube they are able to dilate or constrict this structure. The levator forms a projection on the lateral wall of the pharynx, called

the levator pad, already mentioned. The small m. glosso-palatinus and the broad or palato-pharyngeus runs in the homonymous arches, or pillars of the fauces, and their action is said to prevent any overdistention.

The m. azygos uvulae is attached to the aponeurosis of the hard palate, and is inserted into the uvula, and acts as an elevator and shortener of the uvula.

All these muscles cross and intersect intricately within the velum, and by joint contraction exercise pressure upon the glands, interspersed between their fibers, and thus they also have a mechanical, secretion-expelling function.

Vessels.—The arteries are derived from the external carotid, viz., several branches of the superior thyroid artery. The internal maxillary artery gives off the descending palatine artery, which supplies the soft and hard palate with blood. The facial (external maxillary) artery gives off the ascending (inferior) palatine artery for the velum palatinum and the tonsillar artery for the palatine tonsil. The ascending pharyngeal, a direct branch of the external carotid artery, ascends with it along the lateral pharyngeal wall, and supplies the lateral parts of the pharynx and the Eustachian tube, and also sends branches to the soft palate. The post-nasal space is supplied by branches of the internal maxillary, viz., pharyngea suprema, Vidian artery, and pterygo-palatine.

Of great practical importance is the relation of the palatine tonsils of the neck. (See p. 170.) The *internal carotid artery* runs 1½ cm. behind and a little laterally to the tonsil, separated from the latter by muscles, fat, and loose connective tissue. In the normal course of the artery there is *no danger of injuring the vessel during tonsillotomy;* besides parenchymatous bleeding from the tonsil, *the tonsillar artery,* which is very variable in its origin and course, is more in danger of being injured by the knife. The *external carotid lies 2 cm. from the tonsil;* that is, ½ cm. further off than the internal carotid.

The *veins* form two plexuses—the pharyngeal plexus outside the wall of the pharynx, and the palatine plexus, lying in the soft palate, and which is subdivided into an anterior and posterior plexus. The anterior palatine plexus communicates with the lingual veins, the posterior with the nasal veins. All the veins are finally collected to open into the internal jugular vein. The lymph-vessels show a similar arrangement to the veins. They run to a plexus of lymphatic glands, which are situated high up on the posterior wall of the pharynx, at the bifurcation of the common carotid artery and laterally to the larynx.

Nerves.—The nerve-supply of the pharynx is very complex. The sensory nerves are derived from the pharyngeal plexus, which is formed by the pharyngeal branches of the vagus, glosso-pharyngeal, superior laryngeal, and superior cervical ganglion of the sympathetic nerve; partially, also, from the second division of the trigeminal, which, by the rami palatini, supplies the anterior face of the soft palate.

The motor nerves are derived from various sources, which are still the subjects of disagreement between various authorities. The m. tensor palati is supplied by the third division of the trigeminal (otic ganglion); the rest of the palatine muscles by the facial and spinal accessory nerve; and, according to *Réthi,* by the pharyngeal plexus alone; the latter provides also the motor nerve-supply for the muscles of the pharynx (constrictors and stylopharyngeus muscles).

The secretory nerves are derived from the facial nerve through the chorda tympani.

II. PHYSIOLOGY.

Apart from its insignificant share in the sense of taste, the chief function of the pharynx lies in *respiration, deglutition,* and *phonation,* and it is essentially its complicated muscular apparatus which performs these duties.

In normal, quiet respiration through the nose the current of air passes through the pharynx, whereby the root of the tongue and the soft palate approach each other; in respiration through the mouth the tongue and the velum palatinum separate, the latter approaching more or less closely to the posterior wall of the pharynx (pad of *Passavant*).

In regard to digestion, the pharynx is concerned in the action of swallowing (deglutition). The morsel, masticated and salivated in the mouth, is pushed backwards by the tongue pressing it against the hard palate. Simultaneously, the nasal cavity is shut off by the joint contraction of the levator palati and superior constrictor. (See p. 238.) The necessary closure of the larynx is effected in the following manner: the larynx is lifted upwards and forwards; the base of the tongue is drawn downwards and backwards; and the epiglottis is depressed against the entrance of the larynx. In this way the danger of faulty swallowing is avoided. The swallowed mass, quite immaterial if it be solid or fluid, is, so to speak, syringed with great velocity down into the stomach through a hollow space shut on all sides.

The significance of the pharynx in *phonation* (speaking and singing) has been referred to in the physiology of the oral cavity. (See p. 175.)

The function of the lymphatic ring is still a matter of controversy. According to *Schoenemann*, the tonsils are in their physiological function equivalent to the other peripheral lymphatic glands, and must be regarded as organs of absorption; he considers the pharyngeal tonsil as the most advanced outpost of the lymphatic system of the neck. The enlargement of the pharyngeal tonsil so often seen in children as a product of hyperplasia, but not of inflammation, would then—at least very often—merely answer the purpose of increased function required during childhood. The pharyngeal tonsil, on the other hand, according to *Schoenemann*, may also enlarge without such need on the part of the organism, and here the hyperplasia would be considered as a disease sui generis, requiring operation if it causes trouble.

Brieger, in any case of hyperplasia of the pharyngeal tonsil, sees, from a teleological point of view, a useful institution of the body, "an enlargement of the organ for the purpose of increased function," but the "increased function," in his opinion, is not that of absorption, but, in agreement with *Stöhr*, an increased secretion. This secretory function is not exhausted by the discharge of fluid secretion; but there is, in addition, an abundant production of leukocytes and lymphocytes; the former act as destroyers (phagocytes); the latter as agents, perhaps, of the antibodies (antitoxins) circulating in the blood. That would agree with the fact that the pharyngeal tonsil is at its height of development in childhood, where a protective power is most necessary against bacterial invasion, and that it is subject to involution at a later age, when a certain immunity against infectious diseases peculiar to childhood has been acquired. The enormous regenerative power also of the adenoid tissue, as *Goerke* emphasises, would agree with the assumption of a protective function.

The relapses, which are apparently often met with after radical removal, would thus, according to *Goerke*, not be considered as true relapses, but only as a responsive endeavour of *the lymphoid tissue* to increase according to the need for protection.* But what does not fit in with *Brieger's* theory of the protective function of the pharyngeal tonsil is the fact that the pharyngeal tonsil, as well as the sister organs, the palatine tonsils, very often form the door to pathogenic germs, and that they themselves are very often subject to disease.

With respect to these theories of *Schoenemann* and *Brieger*, therapeutically it would lead to the necessity of leaving a hyperplastic pharyngeal tonsil untouched because of its utility as an organ of secretion or protective absorption, and to operate only if it has grown so large that it causes obstruction or other disturbances (see p. 284); and this standpoint, under those circumstances, seems the only one justified.

Perhaps the tonsils play a certain part in the digestion by moistening the food with their mucous secretion. We leave it an open question whether the leukocytes and lymphocytes emigrated from the tonsils, are the carriers of a saccharising ferment, or whether they form a protective army in the sense of phagocytosis; but in any case we will do well not to overestimate this protective power of the tonsils. Of greater value is the view that the lymphatic ring plays a rôle in the formation of blood. To this points, at least, the histological structure, which is similar to that of the spleen and the lymphatic glands, and also the fact that the lymphatic ring is greatest in children and involutes when the marrow of the bones is able to assume this function after puberty is reached.

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* Translating editor's footnote: An apparent proof of this is the fact that, if a child with the pharyngeal tonsil obstructively hypertrophied, has it removed; and if at that time the palatine tonsils be not enlarged or even visible, then following on the operation, in about three months, the palatine tonsils will usually have become much hypertrophied, frequently so much so as to necessitate removal in their turn.—F. W. F. R.

III. METHODS OF EXAMINATION.

A. EXAMINATION OF THE NASO-PHARYNGEAL SPACE.

Pars Nasalis Pharyngis.—The methods of examination here concerned are posterior rhinoscopy and digital palpation of the naso-pharyngeal space. They have been treated in the first part of this book. (See p. 24, et seq.)

B. EXAMINATION OF THE ORAL PART.

Mesopharynx.—This is performed by pharyngoscopy, inspection, and palpation. For inspection, daylight is sufficient, either used directly, or, better, reflected into the mouth by a reflecting mirror. A thorough examination is, however, mostly only possible by artificial light. In order to get a good view, it will be generally necessary to depress the tongue with a spatula or with the handle of a spoon, etc., which should be done carefully but firmly, and with all due regard to the patient's anxiety or excitability. In spite of everything, it will often happen that the patient retches, a circumstance which complicates the examination, although sometimes it is not unfavourable, because it might then allow of a quick survey. The examination becomes very unpleasant in those cases where the patients are so sensitive that even the simple opening of the mouth causes retching. On the other hand, it is an easy matter with those patients who are able to keep the tongue down flat without any artificial means. In order to inspect and probe the lateral parts, especially the region of the tonsillar sinus, fully, it is necessary to turn the patient's head suitably or to look at it from the side. It will sometimes be necessary to push aside the anterior palatine arch; in the same way a long uvula has to be pushed out of the way by a probe.

In resistant children, who clench their jaws, one can succeed by closing the nostrils, or allowing a third person to do this until the children are forced, by want of breath, to open the mouth; a spatula can then be quickly introduced.

In cases of need, a way may be found by means of the gap behind and between the molar teeth and the jaw.

The pars oralis can be digitally palpated, and also by means of the probe. For the right side of the pharynx introduce the right finger; and for the left side introduce the left finger. In the digital examination of children precautions should be taken to protect the finger from being bitten. (See p. 26.)

C. EXAMINATION OF THE LARYNGEAL PART (HYPOPHARYNX).

The examination of this part also comprises inspection and palpation. Inspection is done in the same way as laryngoscopy, *i. e.*, by the aid of a laryngeal mirror. Usually the inspection of the root of the tongue is combined with this examination. If the tongue is sufficiently pressed down, a part of the epiglottis becomes visible. In adults we can reach, by the introduction of the index finger, usually as far down as to the fifth vertebra. The hyoid bone and the epiglottis can be palpated best from the front. For the lowest part of the pharynx, *von Eicken* and *Gerber* recently described special methods.

The first has devised a so-called laryngeal lever, a firm steel laryngeal probe. Having previously anaesthetized all the parts, the straight proximal portion of the probe is pressed against the upper molar teeth, or should these be wanting, against the corresponding part of the upper jaw, which latter acts as fulcrum; and the distal blunt end being pushed through the vocal cords as far down as the cricoid cartilage or tracheal rings, and the larynx is then drawn forward. (See Fig. 72.)

For the successful performance of hypopharyngoscopy it is important that the anterior muscles of the neck should be relaxed, and this can best be attained by a slight forward inclination of the head. *Gerber* makes the examination, which he calls pharyngo-laryngoscopy, by means of a spatula bent at the forward end to an obtuse angle, the introduction of which is made easier if the patient swallows.

By both these methods the larynx, without any great inconvenience to the patient, is drawn forward from the vertebral column, and the hypopharynx is thus better shown in the mirror than by other methods, which means a considerable gain as regards diagnosis and treatment. One will thus be better able to recognise the ominous cancer of the hypopharynx, and the diverticula of this region, and foreign bodies also, which have not yet entered the gullet, will be the more easily removed; and, lastly, operative manipulations, for instance, incision of an abscess in perichondritis of the cricoid cartilage, removal of a new-growth, etc., can be performed under control of the operator's eye.

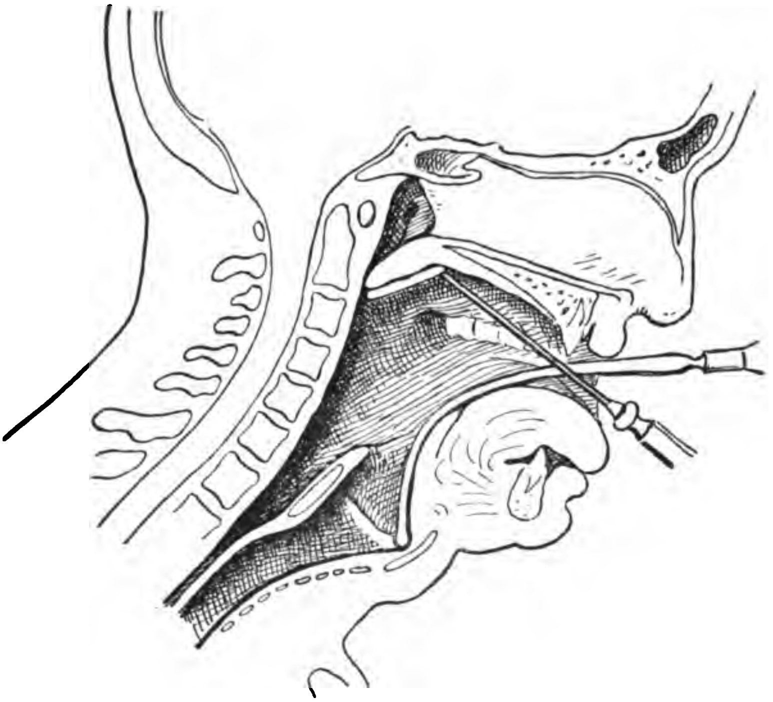

Fig. 72.—Hypopharyngoscopy (after *von Eicken*).

IV. COURSE OF EXAMINATION.

ANAMNESIS.

Many points indicated in the first part of this book (p. 31) are here also to be considered in taking the anamnesis. The subjective symptoms refer not only to the throat, but also to the nose and mouth, as the disease of the throat is very often in *direct connection with that in the nose or mouth.* Therefore the patient naturally complains of the same symptoms, as we have already described, in the respective sections of Parts I and II. We will here mention only the subjective symptoms, which refer to the affections of the throat; these are: general symptoms, disorders of sensibility, of nutrition, and of respiration.

(*a*) **General Symptoms.**—The acute inflammatory processes of the throat are usually accompanied by fever, which may sometimes be very high, or manifest itself by rigors, lassitude, pains in the head and limbs, and loss of appetite. The local appearances do not always correspond with the symptoms, and may be extremely insignificant.

(*b*) **Disorders of sensibility** mostly occur as various forms of pain, *e. g.*, dysphagia. This may be continuous or appears on swallowing food (solid or fluid); it may be still oftener caused by the mere movements of swallowing, and may radiate towards the ear (through the auricular branch of the vagus); in other cases the patients complain of various abnormal sensations, *e. g.*, burning, itching, soreness, feeling of a foreign body, etc. Such sensations, however, would point to neurasthenia or hysteria, if the result of the examination were negative. Nervous or hypochondriacal patients prefer to localise in their throats many of their complaints; but the localisation is usually very indefinite, sometimes even very perverse, and not the least of these is the fear of cancer or consumption; and often syphilitic men, haunted by their fears of syphilis, worry themselves and the physician concerning their throats.

(*c*) **Disorders of Swallowing.**—The patients complain that they cannot swallow, or can swallow only with difficulty, and that the morsel sticks in the throat; or that they swallow the wrong way, which means that part of the food reaches the larynx or nose. The first points to new-growth, affections of the tonsils, or retropharyngeal abscess; and in the latter, to defects of the hard or soft palate, paralysis of the soft palate, bulbar paralysis, or other diseases.

(*d*) **Disturbances of Respiration.**—The cause of obstructed respiration might be found in the naso-pharyngeal space, *e. g.*, adenoid vegetations, tumours, adhesions, etc.; then the patient may complain of nasal obstruction. If the impediment lies in the oral or laryngeal part of the throat (abscess, tonsillar affections, etc.), breathing also through the mouth is obstructed, and the patient complains of want of breath, which, especially in the recumbent position, might amount to a sense of choking. (See also Part I on Oral Respiration and on Disturbances of Voice and Speech. See p. 33.)

STATUS PRAESENS.

1. **Pharynx.**—The examination begins with inspection of the section which is most easily seen, *i. e.*, oral part. At the same time, fetor will be perceived, and we next inspect the nasal part, and lastly the laryngeal part. Examination is completed, if need be, by palpation, and in special cases by a microscopic examination of the secretions or tissues.

(*a*) **Oral Part.**—We look also, en passant, at the anterior portion of the mouth, for in many diseases both regions are affected. The hue or colour of the mucous membrane depends much on the kind of illumination. In daylight or incandescent gaslight the mucous membrane looks lighter, perhaps even a little anaemic. With oil-lamps or electric light it appears more yellowish. The anterior palatine pillars and the uvula are generally brighter than the rest of the pharynx, and the posterior pharyngeal wall, as well as the palatine velum, are usually darker and show greater injection, which contrasts vividly with the lighter coloured neighbouring parts. We must estimate the distance by inspection as to show how far apart are the palatine velum and the posterior wall of the pharynx. If the distance be comparatively large, we think of new-growth in the naso-pharyngeal space or of adenoid vegetations. The region of the palatine tonsil should be carefully examined. (See p. 243.) In order to have a better view of the posterior pharyngeal wall, we may ask the patient to say "e" or "a," which is also a good test for the function of the soft palate.

We must observe the secretions, their quality and quantity, and any alteration of them. *Increased secretion* is found in all kinds of acute and chronic hypertrophic catarrhal inflammations, in inflammation of the tonsils, and in new-growths of the pharynx.

Diminution of secretion occurs in all inflammations accompanied by *atrophy*.

We must note if the secretion is watery, mucous, muco-purulent, or if it has the inclination to dry upon the posterior pharyngeal wall, forming scabs or crusts (particularly in breathing through the mouth, in atrophic processes, in inflammation of the main nasal or its accessory cavities, and when the secretions flow down into the pharynx). (See p. 135.) This is often accompanied by a sensation of dryness, of burning and irritation, causing cough, etc. The nasal part of the pharynx is usually more markedly affected than the oral part. The secretions may be found at times blood-stained in cases of ulceration, tumours, or abscess, and any admixture of soot makes it grey or black.

On inspection of the oral part we must also ascertain if there exists *fetor e pharynge.* In the region of the pharynx, tonsillar concretions, ulcer, or gangrene of the tonsils and naso-pharynx, decomposing tumours, or abscesses are all causes of a foul smell. One must also, of course, look out for possible causes of fetor ex ore (dental caries, affections of mouth and tongue) or for rhinitis foetida. (See p. 33.)

(*b*) **Nasal Part.**—For posterior rhinoscopy and digital examination of the naso-pharyngeal space see p. 24. The mucous membrane of the epipharynx is usually of a brighter red than the oral part.

(*c*) **Laryngeal Part.**—We notice on inspection affections of the base of the tongue and of the introitus laryngis. As to the value of hyopharyngoscopy, we refer to what we have said above. (See p. 244.)

Digital examination must always follow where we cannot get a complete observation of the pharynx, and in other cases, in order to examine the consistency and extension of a tumour or an abscess, or in doubtful cases, to ascertain the size and seat of a foreign body. Occasionally the probe here renders good service.

2. OTHER ORGANS.

A great number of the diseases of the throat are, as has already been mentioned, of secondary nature, following or complicating an affection of the nose or mouth; as it is rare for a disease of the throat to spread into the nose or oral cavity. Most commonly the mouth and throat are affected at one and the same time. In any case the nose should never be forgotten. The ear is often involved in diseases of the naso-pharynx. (With reference to this and the complications of the larynx and trachea see Part I, p. 35, et seq.)

Lastly, we desire to mention the fact that the most varied reflex actions and reflex disorders can be excited in the throat in just the same way as we have recounted concerning the nose.

V. GENERAL TREATMENT.

1. GENERAL MEASURES.

As in diseases of the nose, so in those of the throat, with due consideration for the etiological factors, general treatment will often be the pivot of our medical resources.

In many cases we can try to prevent disease, as, for instance, by hydropathic methods. In other cases climatic treatment in combination with spa cures may be resorted to.

Climatic health resorts, for our purpose, are those places which are free from dust, and are protected against sudden changes of temperature, and are of little relative humidity. For dry catarrh of the throat, on the other hand, a more humid climate, especially the seaside, is very suitable.

Bathing or mineral-water cures should be undergone on the spot, where the climate and hygienic conditions are comparatively best; if this is impossible for some reason or other, the particular "water" may be used at home. The "waters" ought not only to be drunk, but also used for local treatment in the form of inhalations or gargling. The local treatment, if such is necessary, must not be neglected even at a "spa" (bathing place) itself. For the simple and not too long-standing catarrhs of the throat, the alkaline or alkalo-saline springs are most beneficial (Salzbrunn, Ems, Vichy). In the dry affections, "catarrh sec," which are often accompanied by digestive disorders, the saline waters of Homburg, Kissingen, Soden, Wiesbaden, etc., are used, and they act here as solvents and stimulants to the secretions. In the more chronic catarrhs of a congestive nature, the sulphuretted waters are indicated (Eilsen, Weilbach, Aix-les-Bains, and other places), and, in some cases of marked anaemia or chlorosis, the steel springs of Pyrmont, Franzensbad, Schwalbach, St. Moritz, Tarasp. As an after-cure in dry affections, but even also as a primary cure, we may very appropriately resort to the seaside. The taking of "waters" is most suitably combined with inhalations in spraying (drying) houses (Salzungen, Reichenhall, Orb, etc.).

Finally, in the diseases of the throat, as the place where the respiratory tract crosses that of alimentation, special attention should be paid to the quality and quantity of food and drink. Naturally, all irritation, by too hot or spicy condiments and foods or beverages, must be avoided, and alcohol and tobacco cut off; but one must not lose the substance for the shadow in very chronic cases. It is just in these cases that the individual susceptibility for tobacco and alcoholics is very different. A moderate quantity of spirits or one or two mild cigars a day, in many cases, not only do no harm, but would probably, on account of their power of stimulating secretion, be beneficial. "Teetotalisation" of patients often makes hypochondriacs of them.

2. LOCAL TREATMENT.

(*a*) **Applications and Fomentations.**—*Cold Applications.*—For this purpose linen compresses soaked in cold water are used. Wring out the compress well, and between the layers place small pieces of ice, or an ice-bag, or ice collar, both wrapped or enveloped in linen, may be put upon the previously anointed skin. Cold applications are indicated in all forms of acute inflammation, phlegmonous or diphtheric processes, especially at the commencement of the disease, when they will relieve pain. They are materially aided by administering ice internally, which may be given at greater or lesser intervals as lumps to be sucked or swallowed or taken as crushed ice (ice-cream), etc.

Warm Fomentations.—A linen compress, about 4 cm. broad, soaked in cold water and well wrung out, is put round the neck (irregular folds must be avoided), and this is to be protected with a layer of impervious material, such as oil silk, and then covered with a flannel bandage, etc. Some physicians object to the water-proof layer, and cover the cold compress directly with the flannel bandage. Each cover should overlap the underlying layer by 2 cm. (1 inch). The compress can be changed every two or three hours, but need not be taken off during the night. If it causes itching or eczema, it must then be discontinued, or used only at greater intervals, and in the meantime the skin should be dusted with powder.

Irritation of the skin can, in great measure, be prevented by anointing the skin previously to applying the fomentation. The warm fomentations (called, in the form above described, *Priessnitz* cataplasms) act by causing hyperaemia and thus effect a dispersal. Whether the hyperaemia produced acts after the manner of *Bier's* treatment we do not exactly know.

Priessnitz's cataplasms are indicated in all forms of pharyngeal inflammation, in the acute and later stages; and especially if the patient does not tolerate the cold application. As a substitute for these somewhat intricate cataplasms, ready-made bandages or felt pads may be used.

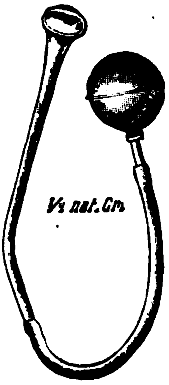

Fig. 73.—Suction apparatus for the tonsil.

Hot Fomentations.—For this kind of fomentation, hot linseed or camomile poultices are applied. They act well as resorbents and to foster suppuration in phlegmonous processes. Cataplasms of hot oil are superfluous.

(*b*) **Treatment by Congestion.**—The use of *Bier's* treatment by inducing hyperaemia has already been spoken of. It has been applied in the various forms of acute inflammation of the throat, particularly in angina and diphtheria, with seemingly good results. For the tonsils, flattened cups, fitted with a tube, are used, and if they are applied once or even several times a day for five to ten minutes at a time, are said to soon relieve the complaints. A final conclusion has not yet been arrived at as to its value; but, any way, it is worth while to try it as a helpful agent in cases of severe angina.

(*c*) **Treatment by Drugs.**—We apply drugs to the mucous membrane of the throat as fluids, in the form of gargles; by brushing, or by irrigation, by massage, or as atomized vapours or sprays for inhalation; or as solid tablets, pastilles, lozenges, or powders.

Gargles, which only come in contact with the anterior surface of the palatine velum, with the tonsils, and perhaps with a small portion also of the posterior pharyngeal wall, may be given in affections of the mesopharynx, especially in the various forms of tonsillitis. In order to convey the gargle thoroughly to the back the patient should recline the head and allow the water to flow backwards, and then, just before a swallowing movement takes place, to quickly spit it out by abruptly throwing the head forwards. Some patients are so skilful as to be able to eject the fluid through the nose.

With regard to the temperature of the gargle, it should be the same as was said of the fomentations; and here experience is often the best teacher. We would, however, warn against overdoing the gargling business, especially in painful affections; also as to using too strong solutions, which ought to be avoided, particularly in children, because of the danger of mis-swallowing.

Douching is done with a specially constructed apparatus through the nose. Its effect concerns the naso-pharyngeal space chiefly. The throat likewise can be douched effectually by means of sprays through the nose, or directly through the mouth. (For the various methods of douching or irrigating, and cautions necessary, see Part I, p. 38.)

Inhalations are, on the whole, better suited for diseases of the larynx than for the throat. Atomised fluids, vapours, or gases are inhaled. If none of the many inhalation apparatus on the market are at hand, one can very suitably improvise such by a jug filled with boiling water and covered by a funnel, towel, or paper bag.

We must not be too sanguine with regard to the therapeutic value of all these measures; for the mucous membrane comes in contact with the drug or solution employed for too short a time, and only within a very limited area. All that we can attain is a certain loosening of the secretions and a cleansing of the mucous membrane, yet this is even sufficient, in many cases, to cure the disease. *The application of drugs is much more effectual* in the dry form, as tablets, pastilles, and lozenges, which are dissolved in the mouth and deposit their medicinal agents directly on to the mucous membrane.

The method of painting or brushing is still better, because of its medicinal as well as its mechanical value; for the same reasons massage is also useful. For painting, brushing, or massage, *Hartmann's* or *Baginsky's* sponge-holder is used (see Fig. 74), and which may be easily inserted into the naso-pharyngeal space. The sponge (pad of wool, swab, etc.) must not be too much saturated, for the fluid is apt to flow down into the larynx.

In catarrhs with copious secretions astringents must be used, and most useful of all is silver nitrate (2 to 5 per cent.). In the dry forms of catarrh, solutions of iodine in glycerine are applied, *e. g.*:

℞. Iodi puri 0.5–1.0
Potass. iodidi 1.0–2.0
Glycerin 30.0
Ol. menth. pip. . . . gtt. ij.

In *acute inflammation, painting is best avoided.*

Powdered drugs* are not often used, and then mostly for the purpose of cauterising. Superfluous caustic is easily removed by gargling with water or wiping with moistened wool. This will be unnecessary, however, if only a little of the caustic material has been used.

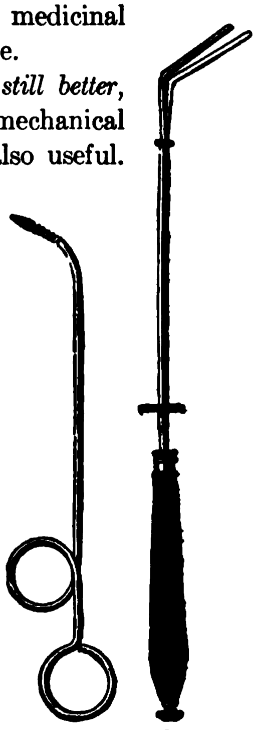

Fig. 74.—Sponge-holder for the throat and naso-pharynx. *a, Hartmann's; b, Baginsky's.*

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* Translating editor's footnote: It is well to warn *against the insufflation of powders*, especially astringent and quasi-caustic powders, as there can hardly be doubt that some cases of acute bronchitis, and indeed pneumonia, following insufflation treatment, have been due to the agent reaching the deeper respiratory passages and setting up irritation followed then by bacterial infection.—F. W. F. R.

(*d*) **Electrical Treatment.**—Under this heading comes the application of the galvanic or faradic current in cases of muscular paralyses or disorders of sensibility.

(*e*) **Operative Treatment.**—With regard to local anaesthesia or general narcosis, the same which has been said for the nose is, ceteris paribus, applicable to the mouth. The application also of the galvano-cautery and of electrolysis follows the same rules and principles (more thereon in the special section).

VI. HYGIENE AND PROPHYLAXIS.

The manifold anatomical and physiological relations between the nose and the throat make it clear that hygienic and prophylactic measures suitable for the one, will be also of much avail as regards the other; at least, so far as general measures are concerned. Therefore, it only remains to refer in brief to the question of "hardening" and air hygiene, to the use of alcoholics and tobacco, etc. The hygiene of the oral cavity, taken all in all, is identical also with that of the pharynx, at any rate of the oral part of the pharynx, which in reality forms an entirety with the mouth.

The cleansing also of the oro-pharynx follows the same lines as that of mouth and teeth. As these latter should be kept clean from early childhood, so should children, as early as possible, be taught to gargle. They learn it soon if they are asked to take a half-mouthful of boiled water and to keep it in the mouth whilst they are told to recline their heads backwards, and at the same time to try and sound a long-drawn "r."

The bad habit of eating or drinking too hot or too spicy food or drinks is often the cause of diseases of the throat. The average temperature should be for beer and hock, 10° to 15° C. (50° to 59° F.); for claret, 18° C. (64.4° F.); for champagne, 8° to 10° C. (46° to 50° F.); water, 12° C. (53° F.); table-waters, 14° C. (57.2° F.); coffee or tea, 40° C. (104° F.); soups, 36° to 50° C. (96.8° to 122° F.); for puddings, roasts, etc., 37° to 42° (98.0° to 107.6° F.).

Generally speaking, all food or drink is injurious which is so hot that it causes burning, or so cold that it produces a sensation of pain in the teeth.

SPECIAL SECTION.

I. MALFORMATIONS AND DEFORMITIES.

Etiology.—Malformations and deformities of the throat are either congenital or acquired. The former represent anomalies in growth (inhibitions of growth); the latter are caused by ulcerative processes (syphilis, diphtheria, tuberculosis, scleroma, burns, or cauterizations, etc.), and are thus characterised by adhesions or strictures.

1. **Abnormalities of Growth.**—To this class belong the clefts in the region of the soft palate, which have been already mentioned among the malformations of the mouth (p. 180). The cleft is often limited to the uvula (uvula bifida). The lower ends of the uvula may, in such cases, be separated from each other or lie so close together that only the probe discovers the cleft between them. Sometimes the cleft reaches as far as the base of the uvula, or it is only faintly indicated by a groove. In other cases the uvula is abnormally small or entirely absent, which latter often points to destructive processes or to the operative zeal of some physicians.

The reverse might also be found; namely, an abnormally long uvula.

In rare cases defects are seen on the soft palate: occasionally on one side only or symmetrically on both sides of the palatine arches, and are oval or slit-shaped. It is doubtful if they are always congenital. The scars which remain after syphilitic or diphtheric ulcers are often so small and indistinct that they are overlooked. I myself observed a case of symmetrical defect of the soft palate, which was unmistakably due to a previous diphtheria.

The tonsils may be rudimentally developed or are absent, or there may be a supernumerary tonsil (tonsilla accessoria), or the tonsil may hang by a pedicle (tonsilla pendula).

Cases of excessive growth are backward prolongation of the septum narium into the naso-pharyngeal space; prolongation of the styloid processes as far as the tonsils; projection of the first two vertebrae to such a degree that the lumen of the naso-pharyngeal space is very much narrowed. Also, atresia of the choanae, mentioned in Part I, would belong to this class.

The congenital (branchial) fistulae of the neck are due to a persistence of the branchial clefts, and are altogether of rare occurrence. They mostly open externally near the sterno-cleido-mastoid muscle, and internally in the region of the tonsils or root of the tongue. Of similar origin are the diverticula of the pharynx. The mucous membrane of the pharynx sometimes bulges out laterally between the muscles, or posteriorly through the posterior wall, forming pocket-shaped or sacciform sinuses, which are known as *diverticula*, and are liable to become very much enlarged by the pressure of respiration or stagnating food. The diverticula seated in the lowest part of the pharynx, and observed mostly in elderly people, the so-called *senile diverticula*, really belong to the oesophagus, and are usually acquired.

Symptoms.—Many of these abnormalities show no symptoms whatever, and may only be discovered by accident. In other cases speech, respiration, and feeding are disturbed. Cleft palate and other defects of the velum palatinum prevent the closure of the naso-pharyngeal space, causing the speech to become nasal (rhinolalia aperta), and particles of food find their way into the nose. A tonsilla pendula irritates the throat and causes coughing and retching. Prolongation of the styloid processes causes disturbances in swallowing. Diverticula, especially those situated lower down, easily become filled with food prone to decomposition, and which causes a sensation of choking. Lateral diverticula are often palpable or visible from outside, and if they are filled with food, they impart the impression of a doughy tumour; if expanded by air, that of a compressible cyst.

Diagnosis.—Many of the anomalies will be recognised on simple inspection; other cases have to be examined by probe, palpation, or posterior rhinoscopy. Sometimes the question will be difficult to decide whether the abnormality present is congenital or acquired. In deeply situated diverticula laryngoscopy or oesophagoscopy will be necessary, but caution is advisable in introducing the bougie, which *Leube*, *Berkhan*, and *M. Schmidt* bend at the fore end. Sometimes diverticula can be felt or seen from the outside (see above).

Prognosis.—The prognosis with regard to restitution ad integrum depends on the seat and the size of the abnormality; quoad vitam, it is always favourable, save from cleft palate in infants, and in rare cases of perforated diverticula.

Treatment.—Some of the said anomalies are a veritable noli me tangere; others, such as cleft palate, have to be treated according to surgical rule. Pendulous tonsils are seized with forceps and cut off, and likewise an excessively long uvula, which causes great irritation. A prolonged styloid process which can be felt as an elastic body from behind the tonsil can be broken by digital pressure from within outwards, or after excision of the tonsil can be dissected out with a blunt instrument and then cut off. The larger diverticula which are visible from the outside may be excised from the outside; sometimes the patients learn by themselves how to empty the diverticula by massage from below upwards. Small diverticula may be cauterised or atrophied by the galvano-cautery.

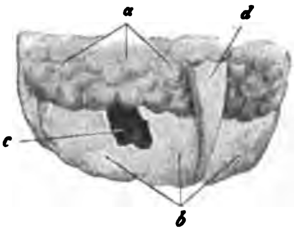

Fig. 75.—Stricture of pharynx (after *Schech*); membranous band-shaped adhesions of the tongue to the posterior wall of pharynx.

a, Root of the tongue; *b*, membrane; *c*, opening in the membrane; *d*, band-shaped adhesion of the back of the tongue to the oral part of pharynx.

2. **Adhesions and Strictures.**—Adhesions occur in various parts of the throat, most frequently in the naso-pharyngeal space, but also in the lower regions of the throat.

Symptoms.—The soft palate is the part chiefly concerned. It may have united partially or wholly with the pharyngeal wall, either on one side or bilaterally; usually the palato-pharyngeal arches are firmly attached to the posterior pharyngeal wall, so that in marked cases the oral part of the pharynx is completely shut off from the naso-pharyngeal space. In other cases sinuses or folds or strictures are formed by the retraction of scars, which alter, more or less, the shape of the soft palate.

In one of my cases the right palato-pharyngeal arch was entirely lost through post-diphtheritic formation of scars. (See Fig. 75.)

In the lower part of the pharynx the fibrous bands usually extend from behind or laterally to the root of the tongue or to the epiglottis. In the latter case they may cover the aditus ad laryngem, leaving only a small aperture (stricture of the pharynx). The inconvenience depends much on the extent of the adhesions. Small adhesions in the naso-pharyngeal space cause no trouble at all, or betray themselves only by a slight nasal intonation. In larger adhesions the symptoms of nasal obstruction or of impeded nasal respiration become obvious. (See Part I, p. 32.) The ear is very often implicated, especially in occlusion by scars of the pharyngeal opening of the Eustachian tube.

Strictures or adhesions in the lower part of the pharynx cause trouble in swallowing, to which disturbances of respiration may be superadded.

Diagnosis.—The diagnosis offers no difficulties in pronounced cases, though examination—possibly hypopharyngoscopy—has to be often repeated in order to get a clear idea of the morbid condition.

Prognosis.—Depends on the site and circumference of the adhesions, and is often unfavourable, for the reason that artificially dilated fibrous strictures show a great inclination to recur in spite of careful and long-continued treatment.

Treatment.—Slight adhesions do not require treatment. More extensive or total adhesions justify operation only if the suffering caused by them is great enough. Sometimes separation of the adhesions by means of knife or scissors succeeds; in other cases the palatine velum has to be carefully separated, and to as far an extent as possible from the posterior wall, and then be kept separated by the insertion of a bougie or repeated dilatations. The operation can be facilitated by inserting a blunt probe through the nose into the naso-pharynx, and then pushing or bulging downwards the fibrous band. Then the depressed dome produced by the probe is incised, and the incision can then be easily enlarged by lateral cuts. Reunion must be prevented, according to *Hajek* and *M. Schmidt,* by systematically stretching the edges of the wounds.

In the lower part of the pharynx adhesions should be severed, under the guidance of the laryngoscope, by means of a blunt-pointed knife, double curette, or galvano-cautery, and readherence prevented by specially constructed tube-shaped dilators (after *v. Schrötter*, *Michael*, and *Jacobson*). Hypopharyngoscopy often simplifies matters, and the disposition to recurrence of adhesions is, on the whole, much less in the lower reaches of the pharynx than in the upper nasal parts.

APPENDIX.

INJURIES TO THE THROAT.

The pharynx is not often exposed to injury, because of its protected situation; the most common injuries occur through unintentional swallowing of a foreign body. (See p. 318.) Would-be suicides usually cut themselves at the lowest part of the pharynx, just below the hyoid bone; still oftener they hit the larynx or trachea. The large vessels are frequently missed, for the suicidal essayists nearly always cut from in front, while they hold the head retracted.

Sometimes the pharynx suffers injury from operation, particularly by the long and unguarded handling of instruments in a nervous, fidgety patient.

Prognosis.—Depends on the nature and site of the injury and on the complications (septic fever).

Treatment.—In slight cases the expectant methods are justified. Ice to suck and swallow and disinfectant gargles, etc., are ordered. For serious injuries, see text-books on surgery.

II. PHARYNGITIS ACUTA (ACUTE PHARYNGITIS).

The division of the acute inflammations of the pharyngeal mucous membrane offers the same difficulties as the same diseases of the mouth. Processes of the same etiology show clinically the most diverse pictures, and their course also varies as to the region in which they are seated, and whether the superficial or deeper structures are affected. I have chosen, for the sake of greater clearness, the same principle of classification as for the diseases of the mouth.

1. PHARYNGITIS ACUTA CATARRHALIS (ACUTE CATARRHAL PHARYNGITIS; CATARRHAL ANGINA).

Etiology.—The causes of acute catarrh of the pharynx are, on the whole, the same as of acute catarrhal rhinitis. The diseases of the nose are, as we know, in intimate connection with those of the throat; acute coryza very often begins in the postnasal space, or soon spreads into the latter from the nose. In acute rhinitis, as well as in acute pharyngitis, bacteria are the main cause of the disease, and are chiefly streptococci and staphylococci; apart from those other causes, such as thermic, chemical, and mechanical irritation, which play a not insignificant rôle. In some cases decayed teeth, and perhaps even dentition, may give rise to inflammation (dental angina).

Children and young persons are very liable to get pharyngitis, because of their lymphatic apparatus being more easily upset; so, also, are weak or exhausted persons, and one could also speak of an individual disposition of persons who suffer once or several times in the year from angina.

The common belief that pharyngitis occurs oftener in the colder seasons does not appear to be based on fact, for it is just in the warm seasons of the year, especially of continuous dry weather, that we observe actual epidemics of pharyngeal inflammations, and which could only be explained by the inhalation of greater quantities of dust, the carriers of bacilli and source of microbes. In pure air, free from dust, as on high mountains, angina rarely occurs. The infectious character of the disease is also marked by its endemic or epidemic occurrences, by the more or less severe disturbance of the general health, and, lastly, by the relation of angina to a whole series of infectious diseases (scarlatina, typhoid, polyarthritis, and certain erythematous and purpuric diseases, etc.).

Equally is angina due to the invasion of bacteria, which supervene after intranasal operations and the use of the galvanocautery. (See p. 80.)

Several French authors of the present time point out that tonsillar angina happens frequently to one and the same person (*habitual angina*), and might be due to the inefficiency of the thyroid gland (*hypothyroidism*). According to this theory, one would have to consider the thyroid gland as a regulating factor of the antibodies or ferments produced by the organism for defence against infection.

Symptoms and Course.—The subjective and objective symptoms are different in proportion to the extent of the inflammation, that is, if the inflammatory process has affected the whole or only a part of the pharyngeal mucous membrane. It must, however, be admitted that the border-line between the healthy and the affected area is never distinct.

(*a*) **Rhinopharyngitis Acuta or Pharyngitis Retronasalis (Retronasal Pharyngitis; Acute Catarrh of the Nasopharyngeal Space).**—Of all the forms of pharyngitis, this disease is the most frequent in association with acute rhinitis. The mucous membrane is covered with a copious secretion, under which it appears red and swollen after some trouble in removing the secretions. If the pharyngeal tonsil is chiefly affected (pharyngotonsillitis), similar signs will be found as in acute palatotonsillitis; *i. e.*, besides a simple catarrh, a *follicular* inflammation occurs, in which whitish nodules or small ulcers due to the sloughing of the lymphatic follicles can be seen; and there is also a lacunar form, which makes its appearance if the crypts, sinuses, and lacunae are affected, leading to the formation of yellowish-white calculi or concretions, which project from these lacunae and are often confluent.

Lacunar angina of the pharyngeal tonsil (angina pharyngea lacunaris seu pharyngotonsillitis lacunaris) is certainly more common than is believed. It is owing to the greater difficulties of posterior rhinoscopy in the cases of acute inflammation that it so often escapes notice.

Perhaps some cases of glandular fever with no clear etiology might have their origin in such a lacunar tonsillitis (see p. 227). All the symptoms may be concealed by a concomitant acute rhinitis.

(*b*) **Pharyngitis Acuta (Acute Pharyngitis; "Sore Throat").**—Acute pharyngitis is the name of the inflammation of the mucous membrane of the oral part of the pharynx, viz., of the posterior pharyngeal wall and posterior surface of the velum palatinum.

The mucous membrane here also is red and swollen in varying degree, and the secretions are at first scanty and viscous, but later on become more abundant, fluid, glairy, or muco-purulent. If the soft palate is affected, it contrasts markedly, by its dilated and tortuous vessels, with the hard palate. Sometimes the lateral walls are chiefly concerned (*pharyngitis lateralis*), or the follicles themselves or the perifollicular area is affected. In these cases one perceives bright-red islands, and within them small losses of substance, *e. g.*, erosions or ulcers, caused by the destruction of the mucous glands and follicles. The uvula, if involved, becomes oedematous.

(*c*) **Tonsillitis Acuta (Acute Tonsillitis; Angina Tonsillaris).**—One distinguishes—(1) *simple catarrhal*, (2) *follicular*, and (3) *lacunar* tonsillitis; if there is present, as the case may be, a simple general reddening and swelling or an affection of the follicles or of the lacunae.

(1) In *simple catarrh*, at first mostly unilateral, the tonsils are covered with secretion, swollen and reddened, as is also the adjacent mucous membrane of the soft palate and its arches.

(2) In *follicular angina* one may notice, besides the signs of simple catarrh, yellowish-white specks and points, scattered over the whole surface, or arranged more closely together, or confluent, corresponding to the suppurating follicles still covered by mucous membrane, and so forming superficial ulcers.

(3) In *lacunar angina* yellowish-white concretions or accumulations are found, which originate in the lacunae and consist of leukocytes, epithelial cells, bacteria, and débris. They project from the crypts and often unite on the surface, forming a kind of pseudomembrane, which, however, has nothing to do with diphtheric or fibrinous membranes.

Diphtheria and lacunar tonsillitis are two quite distinct diseases, which often, however, on account of their resemblance, can only be distinguished by the discovery, microscopically, of *Löffler's* bacilli.

It may be doubted whether we are justified in distinguishing these three forms of tonsillitis, and especially in classifying lacunar tonsillitis (the so-called streptococcal angina) on a separate plane, and so treat the latter as a specific infectious disease. It is probably more accurate to treat them as one disease, different only in the kind of infection, chiefly by reason of the streptococci, but also by staphylococci and pneumococci. The difference in the symptoms and complications (arthritis, muscular rheumatism, erythema, purpura, endocarditis, osteomyelitis, and pyaemia) are dependent on the quantity, quality, and virulence of the bacteria and their toxins.

Even a simple angina, with slight injection of the tonsils, under particular circumstances, may assume a pyaemic character (rigors, arthritic pains, etc.), and it is certain that lacunar tonsillitis is very often followed by complications. Hence we shall not make a mistake if we consider this particular form of tonsillitis, and perhaps also the other forms, as the result of a local infection by pyogenic microbes, which, of course, is very prone to implicate the whole organism by way of the circulation.

Symptoms.—General symptoms are never missing where an infection is the cause. The fever might be low, and only persist for one or two days, but it may, particularly in children, reach a high degree, or begin with rigors and be followed by convulsions, all depending very much on eventual complications. Individual predisposition is a very strong factor. There are people who show a high degree of fever for a minimal affection of the tonsils, and others who, in the severest attacks of tonsillitis, suffer from only slight fever and general disturbance.

In cases of high fever prostration is very pronounced, and is, moreover, complicated by pains in the head or limbs.

The *subjective symptoms* generally depend much on the seat and extent of the disease. Pharyngeal tonsillitis (acute retronasal catarrh) causes accumulation of mucus in the throat and nose, nasal obstruction, and renders speech and swallowing difficult; whereas, in acute palatotonsillitis pain on swallowing, radiating often to the ear, is most pronounced, particularly at the mere attempt at deglutition. The submaxillary glands are tender, the voice raucous and muffled, and there may be difficulty in breathing if the tonsils are much swollen, especially in children.

Complications are, on the whole, not frequent, but it may be often observed that an acute process of the naso-pharynx encroaches on the region of the Eustachian tube and the middle ear, which is manifested by tinnitus, difficulty of hearing, or, in severe cases, by agonising pain in the region of the ear and by discharge from it. In some cases of streptococcal infection metastases in other organs have been noted (see above), and inflammation of the palatine tonsils may spread into the peritonsillar tissue, giving rise to a peritonsillar abscess. (See p. 266.)

The course is determined by the complications, and if not complicated, the cases take from five to eight days to recover.

Diagnosis.—The objective and subjective symptoms are mostly unmistakable. If the symptoms are not very pronounced, an affection of the pharyngeal tonsil should be remembered. Of great importance is the differential diagnosis between lacunar tonsillitis and diphtheria. There are cases, especially in children, where the distinction is very difficult, and even a microscopical examination does not always serve to elucidate matters, and we are then surprised if a subsequent paralysis reveals the true nature of an angina, which, in the first place, we considered harmless.

It is perhaps best to treat any doubtful case of lacunar tonsillitis as if it were true diphtheria, and thus we protect ourselves against surprise and reproach. In general we might treat any case as diphtheria, even if microscopical examination is negative, where the fur or coat is dirty and slimy, and spreads from the tonsils onto the neighbouring tissues, and in which, from the onset, severe symptoms, such as high fever, small and frequent pulse, and prostration, etc., appear.

Prognosis is mostly favourable, but, after all, we should not be rash in foretelling an absolutely favourable course, because severe complications might occur at any time, even when, at first, the disease was slight, for a diphtheria is not infrequently concealed throughout.

Treatment.—Copious diaphoresis sometimes succeeds in arresting or, at least, in shortening an attack of acute tonsillitis. This is done by means of hot drinks and hot packing the whole body. Usually, however, we are called in too late. Aspirin, salipyrin, phenacetin, pyramidon, etc., are very useful at the beginning, and so, also, may be a purgative.

Ice, at first, applied internally and externally, and later on, *Priessnitz's* cataplasms, ought to be administered. For gargles, I prefer the liquor of aluminium acetate, one or two teaspoonfuls in a tumbler of cold or tepid water, or a simple solution of common salt, one teaspoonful to a glass of water. I also prefer to prescribe formamint tablets, every one to two hours one tablet, and menthol lozenges (four or five daily), for the pains on swallowing, or *Avelli's* angina pastilles, which consist of antipyrin and cocaine (caution with respect to antipyrin rashes!).

In scanty or viscous secretion a solution of boric acid in glycerine is very beneficial (acid. boracic., 20.00; ad glycerin., 200.00; a tablespoonful to a glass of cold water).

In the milder cases and in children, besides general systemic treatment, fomentations and gargles will be all that are required. Food must be, at least at the beginning of the disease, cold and fluid.

Complications must be treated as the case may require. Prophylaxis should be directed to a reasonable hardening, in order to reduce the disposition to tonsillitis. If the angina shows a tendency to recur, removal of the enlarged tonsils is justified, though it does not always grant absolute immunity. In general, one should delay any tonsillotomy in acute angina, as with all operations, until the inflammation has entirely subsided, for there is danger of exacerbating by again stirring up the germs of the disease, which were perhaps already declining. Severe cases of lacunar angina should be isolated.

2. PHARYNGITIS PHLEGMONOSA (PHLEGMONOUS PHARYNGITIS; CELLULITIS OF THE PHARYNX.)

Etiology and Pathology.—The seat of a phlegmonous inflammation may be the mucous membrane, but it is chiefly the submucous tissue. All parts of the pharynx may be affected, but the seat of predilection, however, is the loose connective tissue around the tonsil, or in the retropharyngeal tissue. Less frequently the tonsils themselves are affected, resulting in tonsillar abscesses. *Senator* has described a form of infectious phlegmon and erysipelas of the pharynx which are closely related to each other in regard to etiology, and are differentiated only, perhaps, by the varying virulence of their germs.

All these cases are caused by an infection with pyogenic bacteria, encouraged or favoured by carious teeth, crypts, or concretions of the tonsil or by intranasal or postnasal operations. This might help to explain the tendency of the phlegmon to recur, which is so conspicuous a feature in peritonsillar abscess. There cannot be any doubt but that an abscess of the tonsil might cause a peritonsillitis; usually, however, the inflammation is primarily established in the peritonsillar tissues.

The lymphatic glands in the prevertebral connective tissue at the level of the second and third vertebrae, and which begin to atrophy about the fifth year, play an important part in the etiology of the retropharyngeal abscess observed sometimes in infants. The glands are attacked by various microbes and become inflamed and swollen, like a bubo (retropharyngeal lymphadenitis). Apart from this so-called *primary* or *idiopathic retropharyngeal abscess*, there comes under observation a *secondary* form of abscess, which has its origin in some tubercular or syphilitic spondylitis.

Erysipelas of the throat is caused either through the direct invasion of the cocci of erysipelas into lesions of the pharyngeal mucous membrane, or by contact infection from the face; but it is very common for erysipelas of the pharynx to migrate through the nose or mouth outwards onto the face.

Symptoms.—The clinical picture varies according to the seat and extent of the process.

(*a*) **Tonsillar Abscess (Tonsillitis seu Angina Phlegmonosa).**—Abscess of the tonsil is rare—at least much rarer than peritonsillar abscess, and is mostly unilateral. The tonsil is enlarged, swollen, and reddened, and shows more or less distinct fluctuation, with often the same deposits as in follicular or lacunar tonsillitis. The uvula is dragged to one side, or, if both tonsils are affected, behind or in front of the narrowed isthmus. The adjacent tissue often escapes implication. The subjective symptoms are the same as in follicular or lacunar non-suppurative tonsillitis, but are much more severe and pronounced.

(*b*) **Peritonsillar Abscess (Peritonsillitis).**—To what extent the peritonsillar connective tissue of the pharyngeal tonsil is affected often escapes our notice, on account of the difficulties offered to thorough examination by such a case. On the other hand, the peritonsillar tissue of the palatine tonsil is frequently the seat of phlegmonous processes, mostly unilateral and somewhat above and to the outer side of the tonsil, and occasionally also the posterior palatine arch is affected.

The disease begins with severe general symptoms, such as fever, rigors, lassitude, and prostration, to which, sooner or later, pain on swallowing is added, which then radiates towards the ear.

On inspection one sees, at first, only a slight injection of the anterior arch of the palate, while the tonsil itself is normal, and also later on remains itself comparatively unaltered. The patient complains of stiffness, or a feeling of tension, which is due to the great infiltration of the soft tissues; he avoids movements of the head, and keeps it turned towards the affected side, so as to relieve the tension. Opening of the mouth and swallowing are rendered painful, and, finally, the patients are satisfied with merely sipping some fluid. Collections of mucus in the mouth are troublesome and are got rid of with pain; the tongue is thickly coated, and the patients become quickly "run down" and look very miserable.

On inspection, an enormous swelling of the soft palate, particularly of the anterior arch and adjacent parts, is conspicuous. The uvula and part of the palatine velum are oedematous. In some cases the oedema is so marked that the whole affected side looks like a jelly-tumour, blocking the isthmus faucium and thereby causing difficulties in breathing. The tonsil itself is usually only slightly injected and concealed behind the swelling. If the posterior arch is affected, it also bulges forwards.

Inspection is often rendered difficult or even impossible if the patient clenches his jaws, as in trismus. Then we must be satisfied with seeing only that which is possible, even only a small area of bright red and glossy soft palate. In such a case, however, the disease may be recognised, without examination, from the *painful and drawn look, the stiff head, the thick, clumsy speech, in combination with the impossibility of opening the mouth.* (See Fig. 76.)

If the pus is not evacuated artificially, the abscess bursts after a few days, or in some cases even one week, into the mouth or throat. Complications are rare, but migration of the pus, collateral oedema of the head, erosion of the carotid artery with consequent fatal haemorrhage or the formation of a spurious aneurysm, have occurred.

Sometimes the phlegmon encroaches on the neighbouring parts, or spreads externally to the skin or the mouth (angina Ludovici produced by it; see p. 227). The pus having been discharged, recovery quickly takes place.

(*c*) **Retropharyngeal Abscess.**—The onset of this disease is either acute, and is accompanied by fever and pain on deglutition, or it begins more insidiously with symptoms which, especially in children, have no peculiar characteristic. The little patients are restless, hold their head stiffly, and cry when it is moved, refuse food, or swallow the wrong way, so that they cough and fluid regurgitates through the nose. Older children also complain spontaneously or on interrogation of pain on swallowing. These symptoms are soon dominated by signs of impeded respiration. The deeper the seat of the abscess is, the more difficult becomes the breathing. Particularly during sleep, the breathing is very noisy, stertorous, or panting, and in naso-pharyngeal

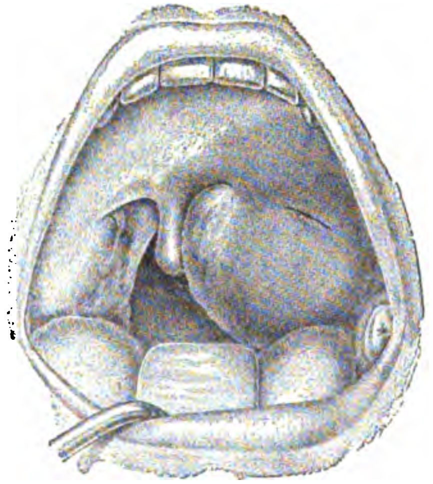

Fig. 76.—Left-sided peritonsillar abscess. Incision.

abscess nose-breathing may be completely obstructed. The signs of dyspnoea appear: cyanosis of the face, swelling of the jugular veins and other vessels of the neck; the wings of the nose move, and the lower ribs are drawn in at each inspiration; and the voice assumes a nasal sound. The lymphatic glands of the neck are also swollen.

In the secondary abscesses stiffness of the neck and tenderness of the spinal processes are very marked.

It is clear that under these circumstances the general health suffers greatly.

The examination is often very difficult in children. Inspection does not permit a full view, especially in deeply seated and small abscess. In such a case palpation is more reliable and will reveal a unilateral, tense, elastic tumour; small abscesses *may*, larger abscesses *always*, fluctuate. In older children or adults the abscess might be inspected by means of the laryngoscope.

If the disease is not recognised and therefore not treated, death from asphyxia may occur, either by the aditus ad laryngem being occluded, by collateral oedema, or by the abscess itself, or it may happen that during sleep the abscess bursts and the pus is aspirated. The increasing debility, especially in the more chronic secondary abscesses, is also a dangerous factor.

(*d*) **Erysipelas Pharyngis (Angina Erysipelatosa; Erysipelas of the Pharynx; Erysipelatous Angina).**—Erysipelas of the pharynx sets in, just as cutaneous erysipelas, with high fever, and to this is added great pain in swallowing. The pharyngeal mucous membrane, and, in particular, that of the fauces, shows a dusky, shiny, varnished appearance, and is oedematous. The process resembles that of the skin; it migrates and affects all neighbouring parts, and if the larynx be attacked, there is great danger of asphyxia through oedema of the glottis. In other cases it migrates on to the outer skin, if it has not originated therefrom. *Sometimes the oedema assumes a bullous character.*

(*e*) **Acute Infectious Phlegmon of the Pharynx (Pharyngitis Acuta Phlegmonosa Infectiosa).**—This disease shows the same features as erysipelas, to which it is very akin. But the fever is usually not so high as in erysipelas, while the mucous membrane is more tense and more swollen. Deglutition is, curiously enough, not so much impeded, but hoarseness and dyspnoea are early prominent, and the great prostration of the patient, passing on into coma, is very alarming. The course is rapid; death ensues—sometimes quite unexpectedly—with signs of heart-failure. The postmortem reveals purulent infiltration of the submucous and deeper tissues, and metastases in other organs and places.

Diagnosis.—*Diagnosis of the Phlegmonous Inflammations.*—Peritonsillar abscess, of all the various forms of phlegmonous pharyngeal inflammation, offers the least difficulties in diagnosis. Its symptoms are very characteristic (see below). An error would be possible only at the commencement, when only a slight injection of the anterior palatine arch is present, perhaps associated with pain on swallowing or a feeling of stiffness on one side.

Whenever such a series of symptoms occurs, one should think, first, of peritonsillar abscess, more especially if the patient reports that he has already suffered from a similar attack, or has been "cut for an abscess in the throat." A linear scar in the region of the soft palate, especially above the anterior arch, mostly points to a previous incision, and will strengthen our suspicion. Diagnosis might be somewhat more difficult if the whole process is somewhat more posterior in situation. But in such a case also, the clinical features are quite characteristic.

Tonsillar abscess is rare. Here the peritonsillar tissue is little affected, whereas in peritonsillitis the tonsil itself shows little or no alteration, if it is at all visible, and is not hidden from inspection by the swelling of the surrounding tissues.

Retropharyngeal abscess, especially in small children, is often overlooked or not recognised. One should, therefore, make it a matter of routine to always palpate the throat in cases where little children are restless at night, snore, refuse food, or misswallow, and to use, if it is necessary for this purpose, a general anaesthetic.

Erysipelas and acute phlegmon are characterised by their rapid course. In the beginning one may doubt whether one has to deal with an acute cellulitis (phlegmon) or erysipelatous angina. If there is high fever from the onset, and should the mucous membrane show the dusky, varnished lustre, and if the process migrates from one side to the other, and perhaps to the larynx, then we are no longer in doubt as to the nature of the disease.

In acute phlegmonous inflammation the local signs are not so pronounced, neither is the fever as high as in erysipelas. On the other hand, the constitutional changes are more predominant, such as prostration, drowsiness, coma, etc.

Prognosis.—The prognosis is usually good in tonsillar and peritonsillar abscess, but might, indeed, become less favourable from intercurrent complications. (See p. 268.)

Retropharyngeal abscess also is amenable to recovery if properly treated, but here the prognosis may be less favourable on account of the primary disease (spondylitis, etc.).

Erysipelas is a very serious disease, and acute infectious phlegmon nearly always ends fatally.

Treatment.—In erysipelas and acute phlegmonous inflammation the general treatment is the most important. Roborants and stimulants are urgently required (strong wines, yolk of egg and wine, ice, fruit-ices, iced milk, etc.). In threatening dyspnoea tracheotomy must be performed.

An abscess of the tonsil or a peritonsillar abscess should be opened. In peritonsillitis, however, we must not incise too early, for we do not, in the beginning, always find pus, though we make our incision on the "typical" place, *i. e.*, 1 cm. above and parallel to the anterior palatine arch. The patient is then disappointed if no pus but blood is discharged, although he may feel relieved from tension, which, however, is not very lasting. Sometimes the abscess will burst through the artificial opening, but that is the exception.

It is better, under those circumstances, to *order warm or hot fomentations and gargles (camomile tea), and not to incise until one has found fluctuation in a definite spot*, which is often situated quite laterally. I never saw any disadvantage arising from this expectant attitude *at the beginning of the disease.*

Wherever it is admissible, a digital examination should be made. In trismus the probe can be used, and by this even, quite a small area of fluctuation may thus be found, as the tenderness is greatest when this area is touched. A double-edged bistoury is used for the incision, and is guarded as far as 1 cm. from the point with cotton-wool or adhesive plaster. The incision should not be too small, and should be afterwards dilated by forceps in order to provide a sufficient exit for the pus. It will sometimes be necessary to slightly reopen the closed wound, on the next day, with a blunt probe, in order to let out any recollection of pus. The after-treatment consists in gargling with warm camomile tea, to which salt or *Burow's* solution (acetate of aluminium) is added. If the swelling does not quickly subside, we must repeat the incisions, for there exists retention somewhere or other. The danger of injuring larger vessels by incising the abscess is not an imaginary one, but should never deter us if we make it a rule to incise only where fluctuation is present. The endeavour to arrest an abscess by ice application is almost always unsuccessful. Hence warm applications are by far the better method of treatment.

Treatment of a retropharyngeal abscess is similar. The guarded knife is introduced under the guidance of the left finger, and a small opening only is at first made, so as not to allow the pus to gush out vehemently; the opening may then be enlarged, while the head of the patient is bent forwards in order to prevent the pus flowing down into the larynx. Here also in some cases we have to reopen the wound on the following day with a blunt probe. Gargles can be ordered for after-treatment in adults.

3. PHARYNGITIS EXSUDATIVA (EXUDATIVE PHARYNGITIS).

Exudative inflammations of the pharynx are to be considered on the same lines as those of the mouth. Very often it is the same process which is localised simultaneously in both parts, or has spread from the one to the other section. (We refer, therefore, to what we have already said on the subject in Part II, p. 186, et seq.)

(a) **Herpes Pharyngis (Angina Herpetica; Herpetiform Pharyngitis).**—*Etiology and Pathology.*—Pharyngeal herpes occurs simultaneously with or following a labial or buccal herpes.

More often it is a disease sui generis. In many cases it is a sort of reflex neurosis, excited by a chill, emotion, or dysmenorrhoea. There are some women who always suffer from herpes of the mouth and pharynx at the commencement of menstruation. Toxic substances also may give rise to an eruption of herpes, as it can be seen in certain infectious diseases or indigestion.

Symptoms.—The symptoms much resemble other forms of herpes. (See p. 186.) The soft palate, the palatine arches, tonsils, and uvula are the most common seat of the eruptions. It may be noted, however, that paresis of the soft palate and a paralysis accompanied by ataxia have been observed after herpetic angina, which certainly points to the effect of some toxic substance.

(b) **Pemphigus.**—This disease may be localised in the pharynx alone, *e. g.*, on the soft palate. Similar eruptions also are mostly seen in the mouth or on the skin. (See p. 187.)

(c) **Aphthae (Pharyngitis Aphthosa).**—Here we also refer to that which we have already said of the same affection of the mouth, for it is very seldom the disease is limited to the pharynx only.

Benign ulcer (ulcus pharyngis benignum) is related to the aphthous ulcer of the pharynx, and is described by *Heryng.* It usually occurs singly and unilaterally on the anterior palatine arch, immediately above the tonsil; the edges are sharply cut, and the floor is coated with a greyish film, which cannot be easily removed. All the symptoms are similar to a slight attack of follicular tonsillitis. The ulcer usually takes from ten to twelve days to heal.

(d) **Other Exudative Processes.**—Occasionally eruptions of urticaria, erythema nodosum, and erythema multiforme have been observed. They form red, slightly elevated patches or papules, usually in association with similar eruptions on the skin, or they precede them. The patients complain of pain on swallowing, salivation, and sometimes dyspnoea.

Diagnosis and Treatment.—As regards these, we refer to what we have already said of exudative stomatitis. (See p. 185, et seq.)

III. PHARYNGITIS CHRONICA.

Etiology.—Chronic pharyngitis originates in repeated acute attacks of catarrh of the pharynx, which may become chronic, especially if they have been neglected. Chronic catarrh is common in persons who are wont to breathe through the mouth, and in those with an abnormally wide nasal cavity; for the mucous membrane of the throat in such cases comes into contact with inspired air, which is not sufficiently warmed previously, and is, moreover, laden with dry dust and bacteria. It is particularly by the foregoing that they explain the occurrence of chronic pharyngeal catarrh in North America. Other causes are the abuse of alcohol and tobacco, the habit of eating too hot or too spicy food, continuous and frequent speaking in the open air or in a dusty and draughty atmosphere, and the aspiration of mechanically or chemically irritating substances (workers' catarrh, as in cotton or wool factories, grinding works, chemical laboratories, etc.).

Chronic pharyngitis often occurs in conjunction with chronic rhinitis, the chronic catarrh having spread from the nose into the throat, or the noxious agents may have affected both regions simultaneously from the commencement. It can be easily understood that the muco-purulent secretions, which run down the posterior nares, adhere to or are retained by the numerous furrows and recesses of the pharyngeal tonsil, and there becoming decomposed, give rise to and maintain an inflammation. Certain constitutional diseases, which diminish, so to speak, the vitality of the body (anaemia, diabetes, tuberculosis, syphilis, gout, etc.), act as kinds of predisposing factors. Diseases of the heart, lungs, and stomach act in the same way by causing chronic hyperaemia or plethora. A certain predisposition to chronic catarrh seems to be inherited, but I venture to say that this so-called inherited predisposition is, for the most part, nothing else than (inherited) lowered vitality, the result of constitutional diseases. This goes to explain how in some families chronic pharyngitis is as firmly established as an heirloom.

The male sex of middle age is far more subject to this disease, for the reason that they are also more exposed to the various noxious agencies causing it.

Symptoms.—Chronic pharyngitis presents various symptoms, all of which are determined by the condition of the mucous membrane and its secretions and by the seat and extent of the disease. Division into different forms of pharyngitis chronica is artificial, for, de facto, they merge one into the other.

Strictly speaking, chronic catarrh is present only where the mucous membrane shows *hypertrophy* (swelling?) and abnormal secretion. Hypertrophy of the mucous membrane later on changes into atrophy, which is then spoken of as *atrophic pharyngitis*, and the secretions may then dry upon the mucous membrane, imparting to it the appearance of a varnished paper—*dry pharyngitis* (*pharyngitis sicca*); this is often accompanied by atrophy of the mucous membrane; but it is also found associated with hypertrophic pharyngitis. *M. Schmidt* maintains that real atrophy of the pharynx only occurs in old age, and that it is often only apparent; that is, if the mucous membrane is less congested in certain places it then contrasts conspicuously with the other hyperaemic or hypertrophic parts, and so appear, by contrast, paler and atrophic in relation to the other more hyperaemic portions.

Chronic pharyngitis, as also does acute pharyngitis, may affect the whole pharyngeal mucous membrane (pharyngitis chronica diffusa, diffuse chronic pharyngitis); or single regions or sections only are affected, and show a different clinical picture.

(*a*) RHINOPHARYNGITIS CHRONICA (CHRONIC RETRONASAL OR NASOPHARYNGEAL CATARRH).

The disease is seldom localised in the naso-pharyngeal space only; the oral part is most commonly affected at the same time. In the post-nasal space it is chiefly the pharyngeal tonsil which is diseased. On inspection, one sees on the posterior wall of the pharynx a dirty grey or greenish muco-purulent secretion, which can be traced up to the roof of the pharynx. The secretion in many cases is derived from the many sinuses of the pharyngeal tonsil, and especially from the recessus pharyngeus medius (bursa pharyngea), and from which it can be seen oozing.*

[**Thornwaldt's* assertion that the middle pharyngeal recess (bursa pharyngea) may be alone affected, independently of other parts, is not very plausible. To assume an isolated bursal pharyngeal catarrh (*Thornwaldt's* disease) is not, therefore, justified.]

The mucous membrane, which may be seen if the mucus is removed, appears swollen, red, or even in a state of cystic degeneration. The tubar pads participate sometimes in the swelling, and betray it by noises in the ear and impaired hearing. The posterior surface of the velum palatinum is red and swollen, and its vessels are dilated; also, the mucous membrane covering the vomer may be found relaxed and congested, and even the posterior ends of the conchae may be swollen and hypertrophied. In a great number of cases the secretion is scanty and dries up, forming scabs, which cover the pharyngeal roof or reach further down along the posterior wall (rhinopharyngitis sicca). This form of pharyngitis is almost always associated with a dry form of rhinitis, and the mucous membrane looks as if atrophied, but really it is oftener hypertrophied. If there also exists a foetid atrophic rhinitis (ozaena), the scabs in the naso-pharyngeal space evince the same unpleasant qualities.

(*b*) PHARYNGITIS CHRONICA (CHRONIC PHARYNGITIS).

This term is reserved for the chronic affections of the oral part of the pharynx, and is analogous to the acute process. It may be doubted whether, as *Schech* states, it occurs oftener here than in other parts of the pharynx; in reality, it is only more frequently seen because of the oral part being more easily and more frequently examined.

The mucous membrane is either diffusely or in isolated patches reddened in varying degrees, and is swollen, looks shiny, and is covered with secretion varying in quantity. The changes are often very slight, as, for instance, in anaemic persons, and contrast conspicuously with the subjective symptoms. In smokers or drinkers, the mucous membrane appears bluish red and congested, and presents dilated and tortuous vessels.

In other cases the lymph-follicles embedded in the pharyngeal mucous membrane are the seat of inflammation. One sees larger or smaller, rounded or oval-shaped prominences on the mucous membrane, of a bright colour, the so-called "granulations" (*pharyngitis granulosa*), which may unite at times to form large excrescences.

Such granulations are also seen in healthy individuals and may not cause the least bad symptoms, and must here be considered as aberrant portions of the lymphatic ring (see p. 237), but they are very conspicuous in children with adenoid growths. Hence the deduction is not to assume pharyngitis always, where such granulations are detected. They are most frequently discovered on the posterior wall, but are found also on the posterior palatine arches and uvula.

(*c*) PHARYNGITIS CHRONICA LATERALIS (LATERAL PHARYNGITIS; HYPERTROPHY OF THE LATERAL WALLS).

On examination of the pharynx one notices at the place where the posterior passes into the lateral wall, on one side, or oftener bilaterally, highly coloured stripes or ridges, which may be followed up to the posterior portion of the torus tubarius. This is a condition of hypertrophy of the salpingo-pharyngeal folds (plicae salpingo-pharyngeae), due to inflammation of the lymphatic follicles, which are embedded in these folds. These hypertrophied folds project markedly, especially during swallowing movements, and their surface has a grained (chagrined) appearance, due to hypertrophy of the numerous "granulations."

(*d*) TONSILLAR CONCRETIONS.

These formations, the significance of which was formerly much underrated, are the products of chronic catarrh in the region of the tonsillar crypts or lacunae. They are composed of migrated leucocytes, epithelial cells, salts of lime and bacteria, and form cheesy, granular, whitish or yellowish, foul-smelling conglomerates, which stick fast in the lacunae, dilating them or projecting from them, and sometimes giving rise to great discomfort.

The tonsils are more or less swollen and reddened; the concretions being themselves a product of inflammation, often act as irritant foreign bodies, and may themselves give rise to hypertrophy or inflammation of the tonsils; or, on the other hand, as the result of the invasion of streptococci, a lacunar tonsillitis or a peritonsillar abscess may arise. It cannot be denied that any toxins formed by the concretions may be passed from them into the circulation.

On the other hand, the tonsils are not necessarily enlarged, and the concretions are often discovered by mere chance, for they may exist for years without causing any trouble at all. These yellowish particles or crumbs are not infrequently only discovered if one searches about in the crypts with a probe; their seat of predilection is usually immediately behind the anterior palatine arch.

Symptoms.—The subjective symptoms do not always depend on the extent of the changes. There are patients who are hardly ever troubled by their catarrh, and others who feel very anxious and complain very much, though they obviously show only slight changes, yet who imagine that they suffer from cancer, consumption, etc. It is a fact that many hypochondriacs are found among patients suffering from chronic pharyngitis.

The *dry catarrhs* generally cause more discomfort than the moist catarrhs. The sensation of dryness is particularly distressing in the morning, when "the whole neck" seems dried up, and the patient has to cough and "hawk" in order to clear his throat from the sticky mucus, which not infrequently chokes, and causes him to retch and vomit (*vomitus matutinus*). *In the moist forms of catarrh* the sensation *of mucoid hypersecretion prevails*, but the patients, however, do not always give a correct account concerning their sensations; and it is remarkable how often perverse sensations figure in the symptomatology of the chronic pharyngitis, namely, such as a feeling of foreign bodies, tickling, burning, itching, soreness, etc.

Concretions in the tonsils often, but not always, betray themselves, especially if one or other of the concretions separates by itself and is discharged; and also by a bad taste in the mouth, of which patients complain. This bad taste may also be accompanied by an evil odour from the mouth (foetor ex ore).

In lateral pharyngitis actual pain may occur, because the inflamed lateral ridges will be squeezed or pressed upon by any contraction of the superior constrictor pharyngis (*e. g.*, as in swallowing). Concretions also may sometimes be responsible for causing pain in the ear and also during deglutition.

Coughing is a very common reflex symptom. It can be easily excited in granular pharyngitis by touching the granules with a probe, or a slight tickling of the base of the tongue and epiglottis by an elongated uvula may cause paroxysmal fits of coughing or choking. At other times the cough is excited by the mucus flowing down into the larynx.

Disorders of voice and speech are very common in chronic pharyngitis. The patients, in particular those who have to use their voice a great deal, frequently complain that they soon get tired of speaking or singing, or that the voice often suddenly alters. The swelling of the mucous membrane, especially of the naso-pharyngeal space, and the relaxation of the muscles, are apt to alter the resonance. The voice loses its pure timbre, and easily becomes tired out, and the more so if it is strained in order to overcome the impediment. An accompanying irritation, or even inflammation, of the larynx contributes much to this result, by which the voice becomes hoarse, or what is called impure or veiled. (See Part IV, Chapter II.)

Complications.—The inflammation of the naso-pharyngeal space sometimes spreads to the *Eustachian tube*, causing obstruction by the swelling of its membranous parts, and also leads by the formation of scabs to inflammation and obstruction of the tube or middle ear. One often sees patients suffering from chronic naso-pharyngeal catarrh who complain of ear troubles, such as noises, fulness, or impaired hearing.

The *larynx* is very often implicated by the descent of the catarrh into it. One finds, especially on the posterior wall of the larynx, the mucous membrane swollen, due to the constant irritation from the downflow of the secretion and, in dry catarrhs, from some dried-up secretion, and scabs on the vocal cords, which tend to alter the voice considerably.

Tonsillar concretions encourage bacterial invasions, which are very prone to eventuate in all sorts of acute inflammations.

Pathology and Anatomy.—The epithelial layers, in particular of the posterior pharyngeal wall, are thickened, so that the mucous membrane looks quite grey at some spots, and the enlarged papillae often extend into the upper epithelial layers. There is augmentation of the connective tissue and a round-celled infiltration about the vessels and glands. The vessels themselves and the glandular ducts are dilated. In granular pharyngitis the lymphatic tissue is hyperplastic and accumulated around the ducts, and the epithelial strata covering the granulations are mostly very thin.

Diagnosis.—The diagnosis should not prove difficult, if one takes into consideration the subjective and objective symptoms. The reports of patients in regard to their sensations and pains must not be taken too literally, for the localisation is not accurate, in some cases even paradoxical. It is sometimes, however, not easy to ascertain whether the secretion accumulated in the upper part of the naso-pharyngeal space accrued from the pharyngeal tonsil or from the nose. If it is possible to trace it *continuously* to the nose, then the secretion must necessarily come, at least to a great extent, from the nose or an accessory cavity. But if there is an area between the choanae and the fornix pharyngis unaffected and *free of secretion,* the latter must then have its origin in the naso-pharyngeal space. *In any case the nose and its accessory cavities should always be subjected to thorough examination.*

Prognosis.—The prognosis as to life is favourable, but as to restoration to a status quo ante, is unfortunately often unfavorable. Restoration to health may, however, take place after a long time. Relapses and exacerbations are very frequent, because the patients do not or cannot take proper care, and in many cases carry their "bit of catarrh" for decades.

Treatment.—Having regard to the great importance of the rôle played by certain hygienic and dietary factors in the etiology and course, we should first of all try to place the patient under the best possible hygienic conditions. Unfortunately, here we often meet with insuperable obstacles in the material position or unchangeable habits of the patient.

Sometimes, however, the noxious trade or profession can be abandoned in favour of a more suitable one. *Tobacco and alcohol* should be forbidden, or, as such an order would hardly be obeyed and is liable to produce hypochondriacs, restraint at least should be inculcated. Our advice must also be directed to the food, voice, etc. (forbidding of too hot or spicy food, abandoning of speaking and singing). Affections of the *nose* and its *appurtenances,* and *constitutional disease,* must be treated.

Local treatment fulfils two objects: removal of secretions and the treatment of the mucous membranes. The secretions are removed, as has already been described in the general section (see p. 252), by washing out through the nose by means of special douches or sprays, usually in the morning and evening, or by gargling or inhalation. Douching does most benefit to the naso-pharyngeal space; gargles best serve the oro-pharyngeal section. In dry catarrhs iodid of potassium (5 : 200, ½ to 1 tablespoonful after meals in warm milk thrice daily) is very useful in order to loosen or dissolve the dried secretions or scabs. In the hypertrophic and sensitive conditions, more astringent applications are suitable, which also reduce the sensitiveness. Generally, weak solutions of sodium chloride or bicarbonate of soda (1 teaspoonful to a tumbler of water) will be sufficient; or a mixture of borax and soda.

℞. Sodii bicarb.	5.00
Sod. biborat.	25.00

M. f. p.

Sig.—One teaspoonful to a tumbler of water, morning and evening, for douching and gargling.

Glycerin is likewise very useful in cases of sensitive patients.

℞. Acid borac.	20.00
Glycerin	200.00

Sig.—A tablespoonful to a tumbler of cold water.

In many watering-places the springs and their salts are taken internally, and are also applied locally. (For the places and springs concerned see p. 250.) I order the water to be drunk thrice daily before meals, and *a small quantity to be gargled.* For douching and inhalation the natural water or its salts are used in certain proportions or concentrations (usually the accompanying measure-glassful to a tumbler of water).

The mucous membrane can be treated by painting or brushing; the diffuse hypertrophic form, with 3–5–10 per cent. solution of nitrate of silver or iodo-glycerin.

℞. Iodi pur.	0.5–1
Potass. iod.	1–2
Glycerin.	30
Ol. menth. pip.	gtt. ij.

The strength of solution and the interval between the sittings (three or four days) depend on the sensitiveness of the mucous membrane.

We must paint the naso-pharyngeal and oro-pharyngeal regions energetically—small haemorrhages do not matter. In some cases, if the hypertrophy is more circumscribed or defined, caustics or the galvano-cautery or cutting instruments must be used.

Granulations found on the posterior wall are only attacked if they are tender to the touch with a probe or cause actual pain. Having cocainised them, one applies lapis infernalis, trichloracetic or chromic acid (see p. 40), or the galvano-cautery, the flat point of which should be bent rectangularly. Larger granulations may be removed with a sharp double curette, like that used for intralaryngeal operations, or they may be cut away with long curved scissors bent on the flat. (See Fig. 77.) Both instruments can be employed for hypertrophic lateral pharyngitis, but if these lateral parts are of moderate size, then caustics or the galvano-cautery may be used.

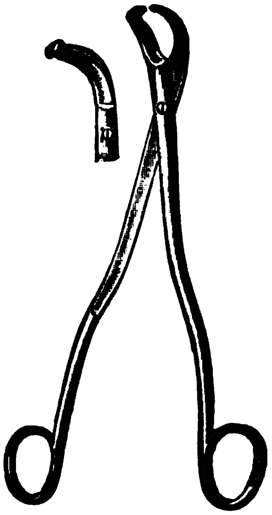

Fig. 77.—Pharyngeal scissors with the blades bent on the flat (after *Cordes*).

It is impossible to remove all the morbid tissues at one sitting, for the reason that it is not advisable to cauterise or cut too much at once, on account of the violent reaction. It is an important point to follow the lateral strips or ridges sufficiently, both upwards and downwards as far as possible; half measures are of no use. On the other hand, one should not proceed too rigidly and schematically, and cut away each small granule and destroy each slight trace of a lateral ridge. Here, more than ever, each case must be treated according to its own peculiarities.

If the pharyngeal tonsil is the seat of disease and the secretion arises from one or more recesses, one achieves the best cure by operation. (See p. 290.) The application of caustics, which has been recommended, is troublesome, and the use of the galvano-cautery not without danger.

The uvula should not be cut away, unless it is so much enlarged that it touches the back of the tongue or the epiglottis, causing coughing or retching. In order to perform uvulotomy or kiotomy, the uvula is seized with the forceps and cut off with *Cooper's* scissors, leaving a little stump 1 cm. in length. Bleeding is insignificant.

After-treatment here, as in other caustic or surgical manipulations, consists in the administration of cold fluid food, ice, and disinfecting gargles for two or three days. Concretions in the tonsils are treated after *Hartmann's* method, by pressing on the tonsil with a squeezer in different directions, especially on the anterior palatine arch. (See Fig. 78.)

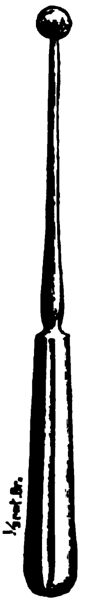

Fig. 78.—Tonsil squeeze (after *Hartmann*).

By this means a sero-purulent fluid or cheesy matter is squeezed out. Should the disposition to form concretions continue after this manoeuvre has been repeated several times, the lacunae in which the concretions are lodged can be split up by an instrument which has the shape of a blunt hook (*M. Schmidt*). The little hook is inserted into the lacunae and the tissue torn downwards; thus the lacunae are changed into open channels. Recently the suction method, after *Bier*, has been applied for the removal of the concretions. In very obstinate cases the concretions, together with the lacunae, can be taken off by *Krause-Heryng's* double curette or *Hartmann's* conchotome, or the tonsils may be resected in toto (tonsillotomy).

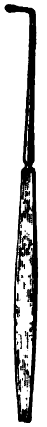

Fig. 79.—Tonsil splitter (after *M. Schmidt*).

IV. VEGETATIONES ADENOIDES.

Etiology.—Adenoid vegetations, the importance of which for adolescents was first pointed out by the Danish physician, *Wilhelm Meyer* (1873–74), occur mostly in childhood, between the fifth and the fifteenth year. During this period the lymphatic tissues, as we have already mentioned, show greater irritability. They are, however, sometimes found in infants, whereas after the twentieth year they are less frequent, decrease in frequency thereafter, and after the thirtieth year are only met with in rare cases, their disappearance corresponding with the physiological retrogression of the pharyngeal tonsil. Statistics of their occurrence are very doubtful; they oscillate between six and 20 per cent.; and exact investigation, according to *Burger*, shows a still larger percentage, viz., 30 per cent.

As regards the causes, there is not yet a clear conception. Climate has no influence, according to my experience. Unfavorable hygienic conditions seem to play a certain rôle in the etiology of the adenoid growths, as is shown by their frequent occurrence among the so-called lower classes. Repeated colds, chills, and nasal catarrhs may lead to hyperplasia of the pharyngeal tonsil, perhaps on account of the invasion of bacteria. In that case the vegetations would be the product of chronic inflammation. On the other hand, many consider them as merely an abnormality of growth or nourishment, *i. e.*, as a genuine homoplastic hyperplasia of the normal organ (*Schoenemann*, see p. 242). Heredity may play its part, as there are families in which, through several generations adenoid vegetations are found. Even within one family, if, for instance, the father has suffered from adenoids, then the children who resemble the father will also suffer.

Pathological Anatomy.—Adenoid vegetations occur in two forms: as a broadly seated, flat or globular, rather hard growth, showing a lobulated surface due to numerous more or less deep fissures (*simple hyperplasia of the pharyngeal tonsils*); or it is of a more soft consistency, showing an accumulation of conical or villiform excrescences (*adenoid vegetations, properly so called*). Both forms are not always distinct, and can merge, the one into the other; their color is greyish red. Histologically, one finds in both forms the same structure as in the normal adenoid tissue, viz., a reticulated connective tissue with round-cells between the meshes, and numerous follicles, the whole covered with ciliated or squamous epithelium; and in older and harder hyperplasias the structural connective tissue is also increased.

Giant-cells and tubercle bacilli, which have been found in the texture of the adenoids, are probably due to latent tuberculosis of the pharyngeal tonsil. The question whether it here reveals a primary infection of the tonsil or a secondary invasion from another quarter of the body still awaits an answer.

Symptoms.—In the forefront of the symptoms stands obstruction of nasal respiration, which is the more marked in direct proportion to the blocking of the posterior nares. The consequences of continuous mouth-breathing are discussed in Part I. (See p. 33.) The permanently open mouth, in conjunction with the shortened upper lip and the relaxed facial muscles, gives the face a vacant or stupid appearance. Children cannot blow the nose, and are restless in bed at night, owing to the larynx being barred by the retracted tongue. They snore or pant and suffer from nightmare (pavor nocturnus), and look very tired and sleepy in consequence of their broken sleep. The voice has the character of rhinolalia clausa. (See p. 33.)

The phonation of the half-consonants is changed, and sometimes stuttering or stammering may be caused. The air-current entering through the mouth is apt to exsiccate the pharyngeal mucous membrane, thus giving rise to catarrh of the throat and larynx; the latter, however, is mostly due to the irritation exercised by the secretions flowing down from the naso-pharyngeal space. The nose may also show catarrhal changes, usually of a hypertrophic, more rarely of an atrophic, nature. The little patients suffer, as it is called, from a "stuffy cold"; the secretion running down from the nose over the upper lip gives rise to eczema of the entrance of the nose or to erosions and swelling of the lip, which circumstance is often mistaken for scrofulosis. Nose-bleeding, which is reported by various authors, did not come under my observation, and is denied by *Zarniko* and *Thost;* I am inclined to attribute it to an artificial bleeding resulting from nose-picking, a habit into which children fall if the nose irritates from eczema of the entrance.

Obstruction of the nose renders respiration superficial, and thereby limits the expansion of the chest, which in due course leads to anaemia and aggravates the disposition to inflammation of the bronchi.

Other consequences or changes associated with them are: deformities of the nose, high vaulted palate, deviation of the septum nasi, and anomalies of the growth and position of the teeth. (See p. 36.) Deformities of the palate and dental caries, however, are not pathognomonic, for some children with adenoid vegetations often show broad, flat palates and also have good teeth.

One of the most frequent complications is disease of the auditory apparatus. Abnormally large vegetations may, by blocking the orifice of the Eustachian tube, cause catarrh of the tube or middle ear, or inflammation of the ear might be produced by the direct extension thereto of the inflammatory processes existing in the naso-pharyngeal space. Even moderate adenoid vegetations form a dangerous focus for the ear close by, and very often *difficulty in hearing in children, is the only symptom pointing to adenoid growths.* Repeated inflammations of the middle ear or periodical difficulty in hearing in childhood should always turn our attention to the naso-pharyngeal space. The *aprosexia* of the authors, that is, inability to direct the child's attention to one point, can be explained in many cases by the impaired hearing. Other authors suggest that aprosexia is due to congestion in the cerebral veins and lymphatic vessels. These children are admittedly inert, lazy, ill-tempered, indisposed to learn, and are backward, and sometimes create the impression that they are stupid. On the other hand, there are many children who, in spite of enormous adenoid growths, are fine scholars.

Lately, "adenoid vegetations" have been described as a reflex exciting organ, for instance, nocturnal enuresis has been brought into connection with it. But it may be questioned whether this connection actually exists or whether the nightly wetting of the bed is only a sequel of a neuropathic condition, produced and kept up by the vegetations. Other neuroses also, such as chorea, asthma, laryngismus, etc., have been attributed more or less correctly to adenoid growths.

Objective symptoms can only be found by posterior rhinoscopy. (See Fig. 80.)

It is important to ascertain whether the enlarged pharyngeal tonsil forms a more diffuse, even enlargement, or is of a more lobulated, fissured, and villous character; whether it is seated in the centre or more laterally, and whether the vomer and choanae are involved; but one must not also allow one's self to be deceived by the image in the mirror, which shows all parts foreshortened. It very often happens that vegetations are larger after removal than one was disposed to assume during examination. In many cases the surface of the growth is covered with fluid or dried secretion and scabs.

Diagnosis.—If we are called upon to examine a child who is supposed to suffer from obstructed nose for a long time, is restless at night, snores, or hears badly at certain times, we at once presuppose adenoid vegetations. And this presupposition will gain in strength if the child also shows the so-called "*adenoid facies,*" viz., stupid look, open mouth, deformities of palate and teeth, etc.

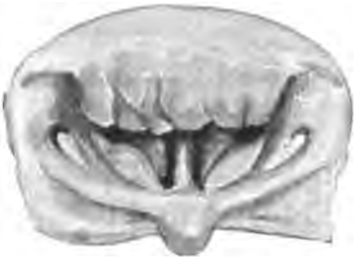

Fig. 80.—Adenoid vegetations.

The appearance, however, may be deceptive. There certainly are children with adenoid vegetations in whom the adenoid habitus is missing, and others who, in spite of the typical adenoid appearance, do not show a trace of adenoid hyperplasia. This circumstance would very well agree with the contention of some authors, according to whom the aforementioned deformities of the skeleton are not in resultant connection with the adenoid growths, but have to be considered together with the latter as a sign of a general physical and psychical degeneration.

To further insure the diagnosis, the voice should be tested as to its sound (nasal), and the oral part of the pharynx inspected, which is not difficult, even in small children. The comparatively great distance between the velum palatinum and posterior wall of the pharynx is at once to be noticed, and one may be able to see solitary or multiple granulations or follicular swellings on the posterior wall, which form quasi-outposts of the adenoid vegetations. Their size is in direct proportion to the adenoid vegetations. In rare cases the latter may reach so far down that they can be seen by inspection from the mouth.

If the anamnesis and external inspection, as well as the pharyngoscopy, should leave us in doubt, we may try anterior rhinoscopy. There is no difficulty in seeing adenoid vegetations from in front if the nose is wide enough. One sees in the depths of the nose, on both sides, more often in one side only, a mass with a smooth or rough surface, usually with convex lower margin, which, on swallowing or intonation of "i" or "u," moves slowly upwards, as can be observed by the reflected light. The probe will prove very useful in some cases, especially in soft, spongy vegetations, in order to ascertain their consistency. Unfortunately, the nose is mostly narrowed or blocked (swelling of the concha, deformities of the septum, polypi, etc.), so that probing and anterior rhinoscopy are impossible. In those cases we assist ourselves by posterior rhinoscopy. Wherever we can, in adults and children, we should try to perform posterior rhinoscopy, because it gives the best result. If this should also fail, palpation must be resorted to. This, however, is a somewhat crude method, and not infrequently causes a little bleeding, making the children still more nervous and resistant, and not giving such exact results as is usually supposed. Indeed, it seems to me that palpation is only sure in cases with larger sized vegetations, which, moreover, betray their presence by other signs.

Differential diagnosis must sometimes be made between vegetations and other tumours of the naso-pharyngeal space, viz., typical naso-pharyngeal polypi (see p. 324), sarcoma, and lymphosarcoma. Consistency and appearance may here be very deceptive, and only a microscopical examination of excised portions will clear up the matter in doubtful cases. *Zarniko* mentions the possibility of mistaking leukaemic infiltrates for adenoids.

Prognosis.—The prognosis in general is favourable, but one should bear in mind that adenoid vegetations constitute a certain danger to various organs and to the entire health. Besides this, the pharyngeal as well as the palatine tonsil must be considered as the chief gate of entrance for the several bacteria of tuberculosis and other infectious diseases. On the other hand, operative removal, if properly performed, has a marvellous effect on the development of children, and in adults also a series of troublesome catarrhal symptoms are often relieved by it. Recurrences, however, may happen.

Treatment.—The one and only reliable means of curing the disease is the removal of the vegetations (adenotomy).

Indications and Contraindications of Adenotomy.—Small adenoids which do not cause trouble and are only discovered perchance may very well be let alone, as they may possibly fulfil a useful function (see p. 242), and later on fall into atrophy by themselves. But if the vegetations are an impediment to respiration or a source of inflammation, or if they cause the patient to suffer from all sorts of catarrhal, nervous, and other complaints, then removal, without regard to the age of the patient, is indicated. The operation is *contraindicated* in acute fever, in severe anaemia, haemorrhagic diathesis, and inflammation of the middle ear in the first or acute stage. On the other hand, in long-standing or suppurative otitis media, adenotomy is beneficial, as well as in catarrh of the Eustachian tube and middle ear.

The following points are of importance for the performance of the operation, which should be done, if possible, on an empty stomach:

(*a*) *Assistance.*—Without proper assistance the operation, which has to be done quickly and energetically, is impossible. An assistant seated on a chair has to keep the patient firmly, as we have already described for the removal of a foreign body. (See p. 110.)

(*b*) *Anaesthesia.*—A good assistant will render anaesthesia superfluous in small manageable children, and also in adults. If the parents especially desire it, and older children if they are very resistant, may have chloroform administered. But the anaesthesia should only be light, so that the reflex of coughing and swallowing should not be abolished, in order that the blood, which might possibly run into the throat, may be coughed up and expectorated.

Ethyl bromide may also be used. (See p. 42.) The anaesthetic is administered to the child in a recumbent or reclining position, and for the purpose of operation the child is made to sit up. In fact, narcosis will only be necessary in obstinate children, if the *pharyngeal and palatine tonsils* have to be removed at the same time, for the sake of both operation and children.

(*c*) *Instruments.*—Of all the numerous instruments, the so-called adenotomes (ring-shaped knives) are most suitable. (See Fig. 81.)

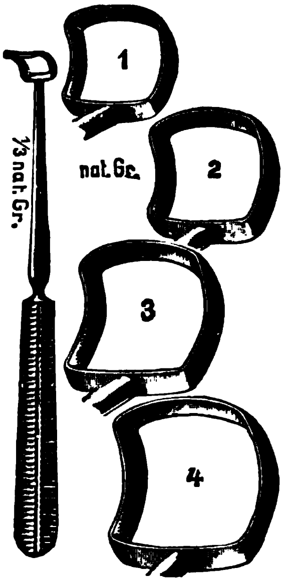

Fig. 81.—Adenotome (after *Beckmann*), in various sizes.

I myself exclusively use *Beckmann's* adenotome, which is a modification of *Gottstein's* nasal curette. As a rule, the largest sized instruments should be employed. The larger the instrument which one is able to introduce, the more radical is the effect of the operation. The tongue is depressed by means of a tongue depressor or firm spatula; the jaws, which are often clenched, must be kept open by a gag. In many cases one will succeed in opening the mouth by compressing the nostrils. For severing any portions of vegetation left behind, *Heyman's* forceps (see Fig. 35) or curved scissors, or a snare, may be used, and, of course, all these instruments must be sterilized and kept handy before operation.

The operation is performed in the following way (see Fig. 82): The operator, with the reflector on his forehead, is seated in front of the patient and introduces *Beckmann's* ring knife behind the soft palate, while the tongue is kept well depressed in such a manner that the handle is directed downwards. The soft palate is pulled *firmly forwards*, and then, by pushing the instrument as far upwards as possible towards the fornix pharyngis, the adenoids are then cut away by sweeping it firmly backwards and downwards. And now, without removing or turning the knife, the same sweeping movements are made laterally to the right and left alternately.

It is necessary to pull forward the soft palate in order to reach the anterior surface of the adenoid growth, and the pushing upwards towards the fornix is required in order not to leave too much behind. The beginner usually makes the mistake of not going sufficiently high. That the adenoid tissue has been cut

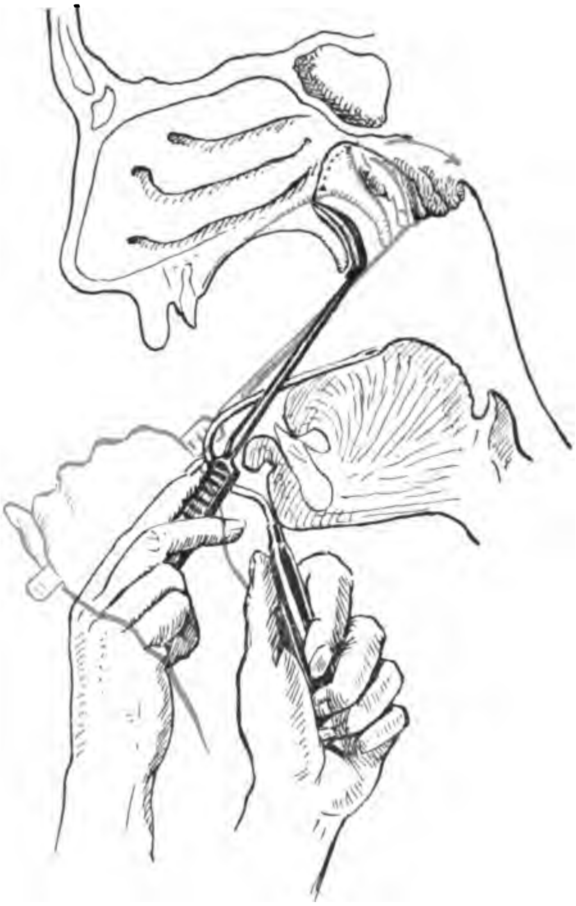

Fig. 82.—Diagrammatic representation of adenotomy. The black drawing shows pulling forward of the velum palatinum by the adenotome inserted behind it. The red drawing shows how the instrument is moved in the direction of the red arrow.

through, one knows by the peculiar crunching sound. One succeeds in many cases in getting the pharyngeal tonsil out in one piece or divided, or it may be coughed out. In other cases the tonsil may be retained through some kind of spasm of the soft palate, and is cast out later through the mouth or nose. The whole operation must be rapidly done. Immediately after the knife is withdrawn the head of the patient is quickly tilted forwards, on account of the considerable haemorrhage which might follow, but this, however, soon ceases. Finally, the patient is requested to blow separately each nostril, if circumstances allow of it, in order to remove all coagula, but this, however, is not urgent.

After-treatment.—The operation being finished, the child is best put into bed for one day, and should be given at first nothing but cold fluids to take, such as milk, lemonade, yolk of egg with sugar, or fruit-ice, or, if the pain is great, lumps of ice to suck, and externally cold fomentations. On the second or third day food may be lukewarm and sloppy. The little patients usually feel all right after their first sleep; all manipulations afterwards in the nose and naso-pharyngeal space are superfluous or even harmful, and should, therefore, be avoided. Children who are able to blow their noses should be told to do so with their fingers (à la paysan) in order not to endanger the ear. Children attending school must be kept away for from four to five days, according to how they progress.

Complications.—(*a*) *During Operation.*—It often happens that a portion of the tonsil is not cut through and still hangs by a thread of tissue swinging or touching the tongue. This is always the case if the patient, after operation, chokes and coughs, and if the haemorrhage has been very severe. Such a piece must be cut away by scissors or snare or may be pinched off by forceps. It must not, however, be *pulled off*, because the posterior wall of the pharynx might be thereby endangered. Haemorrhage usually ceases as the result of this small after-operation. Sometimes the piece cut off has been swallowed during the operation and will be vomited subsequently along with the blood which has also accompanied it.

(*b*) *After the Operation.*—After-bleeding is rare, and should it occur and be severe, tamponing of the naso-pharyngeal space may be necessary. (See p. 67.) Wound infections, provided the operation was properly done, are still more seldom. Some cases of glandular fever are perhaps due to post-operative infection. Of other complications, I may name, from my own experience, a case of wry-neck and two cases of paralysis of the velum palatinum.

The result of the operation manifests itself almost immediately. The children sleep better, the voice loses its peculiar sound, etc. Other symptoms, of course, disappear later. Breathing through the nose still remains impeded for a time, on account of the reactionary swelling, and in certain cases, if the nasal cavity itself is obstructed, it will not subsequently get better. Children frequently have to be taught again how to breathe through the nose; and they must be taught by systematic exercises to breathe through the nose with the mouth shut, by strict instructions, eventually by binding up the jaw during the night.

Complications or consequences which are already present before the operation, and do not subside by themselves after it, must be treated according to rule. Relapses or remaining portions require a repetition of the adenotomy, provided that they cause trouble; or if the nose is wide enough and the patient allows it, they can be removed with the snare from within the nose.

V. HYPERPLASIA TONSILLARUM (ENLARGED TONSILS; HYPERTROPHY OF THE TONSILS).

Etiology.—The palatine, like the pharyngeal, tonsil in childhood, especially if there exists a *lymphatic tendency*, is often subject to hyperplastic changes. Heredity seems also to play a causal rôle, and repeated catarrhs are very apt to produce tonsillar hypertrophy.

Pathological Anatomy.—The enlargement of the palatine tonsil having become stationary, may be considered as a product of chronic inflammation of its entire structure. If the adenoid tissue is chiefly concerned, the enlarged tonsil is soft and of a red or dirty pink colour; but if the connective tissue is in excess, as is mostly found in adults after repeated attacks of angina, the consistency is harder, the tonsil is pale, showing on the surface a network of white fibres, and between which the dilated openings of the lacunae are visible. In such cases the tonsil shows a fissured surface, and is often adherent to the palatine arch. Concretions are often found, and their importance with regard to tonsillar hypertrophy has been already described (see p. 277); but it might also be possible for the concretion to have been produced at a later stage in the dilated lacunae.

Symptoms.—The symptoms are, for the most part, the same as those in adenoid vegetations, which, as we have said, are very often associated in childhood with tonsillar hypertrophy. In cases of very enlarged tonsils the impeded breathing may become so aggravated as to amount to dyspnoea. Children are restless and snore during sleep at night; the voice is guttural and thick, the enlarged masses impede the free display of the soft palate, and fluid will sometimes regurgitate through the nose.

On examination we find both tonsils, as a rule, swollen, the one sometimes more than the other, and the enlargement may reach as far as the middle line or exceed it. The tonsils project still further forwards during movements of swallowing, which admits of an estimation as to how far backwards the hypertrophy reaches. In some cases only single portions of the tonsils are enlarged; the surface then appears uneven and lumpy, particularly if it has already been the subject of operation. The posterior pharyngeal wall, as far as it is visible, also shows accumulations of follicles (granulations); the lateral folds (bands) may be hyperplastic. The glands in the neck are often swollen as a consequence of several previous attacks of angina.

Diagnosis.—The diagnosis never offers any difficulty, but one should bear in mind that syphilis, which is so prone to attack the lymphatic tissue, likewise produces hyperplasia of the tonsil. It may also be mentioned that leukaemia may cause tonsillar hypertrophy.

Prognosis.—The prognosis, apart from the leukaemic form, is always good. We must not forget, however, that the enlarged tonsil, with its unevennesses and crypts, is a good nidus for bacteria, and is, so to speak, the breeding-ground of angina.

There is, moreover, quite a large number of people who, in spite of tonsillotomy, are subject to repeated attacks of angina, with or without concretions in the stumps which are left.

Treatment.—If the tonsils are so large that they form an obstacle to respiration, or if they have been repeatedly the seat of inflammation and concretions, operative removal, viz., tonsillotomy, is justifiable. This can be quickest done with a tonsillotome. I prefer the instrument of *Mathieu-Fahnenstock*, which I possess in three sizes. (See Figs. 83 and 84.) While the tongue is depressed, the oval or ring-shaped knife is *slipped as far as possible over the tonsil;* the adjacent structures (arches) must be pushed aside, and care should also be taken that the lower edge of the tonsil is well within the ring. Then the sliding ring-knife is firmly pulled forwards. By this manoeuvre the tonsil is transfixed by the harpoon of the instrument, and the tonsil cut off by the ring-shaped knife, by as much as had projected beyond the palatine arches. (See Fig. 84.)

With a little practice a double tonsillotomy can be completed in a few seconds. Should the tonsil be much fissured and soft or concealed, a polypus snare may occasionally be used. Some advise that the tonsil be pressed from outside towards the median line; *this seems to me superfluous, and not without danger* with regard to the large vessels in this region.

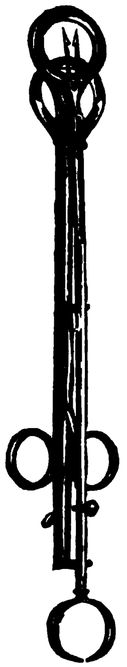

Fig. 83.—Tonsillotome (after *Mathieu-Fahnenstock*).

Complications During and After Operation.—(*a*) *Bleeding.*—The most unpleasant, but luckily rare, complication is the injuring of a large vessel. Experience shows that bleeding is greater in the hard tonsils of adults, because the vessels embedded in the hard fibrous tissue do not retract and are the easier kept gaping. Injury also to the palatine arch may cause bleeding. Haemorrhage sometimes occurs several hours after the operation.

The best treatment for severe haemorrhage is digital compression of the bleeding spot. For this object the finger is armed with cotton-wool, which may be dipped in perchlorid of iron or peroxid of hydrogen. The ordinary styptics are unreliable. *Proebsting* has constructed a special form of pressure forceps for tonsillar haemorrhage. Sewing together the palatine arches, transfixion and torsion of the bleeding site, are all complicated procedures. Ligature of the respective carotid artery would have to be considered as an ultimum refugium.* It has already been done in several cases, and was not followed by any untoward result. Slight bleeding ceases of itself if rest and ice are applied. Tonsillotomy should be avoided in haemophilia, and destruction by the galvano-cautery would be the best substitute in such a case. *M. Schmidt* advises the use of the galvano-cautery wire snare.

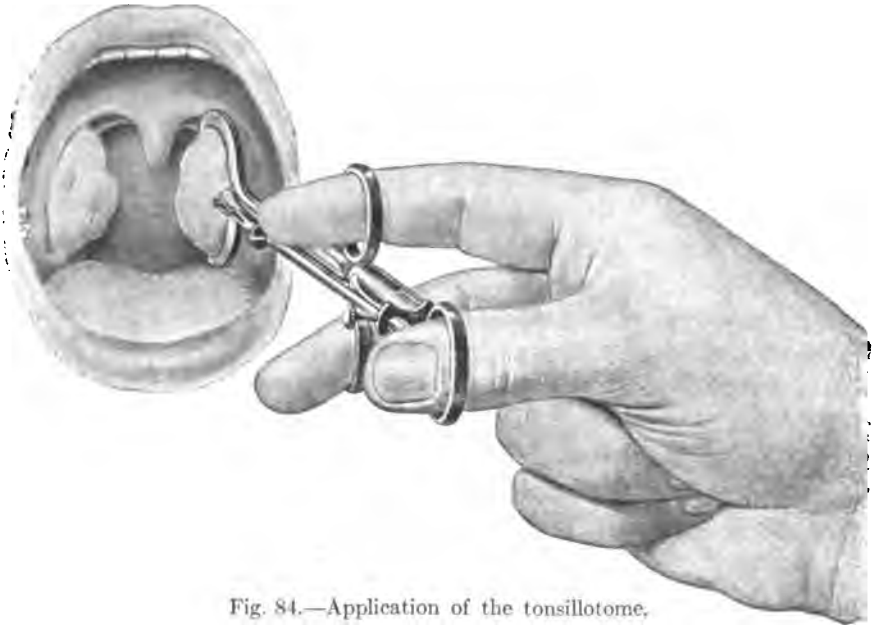

Fig. 84.—Application of the tonsillotome.

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* Translating editor's footnote: It would appear from experience that if half a tonsil be shaved off, the stump is more prone to bleed than the stump of a three-quarter or seven-eighths removal. This being the case, if haemorrhage proves troublesome, it has been known to at once cease by removing a further portion of the bleeding tonsil by guillotine or punch forceps; or the bleeding stump may be evulsed; the bleeding then soon ceases.—F. W. F. R.

(*b*) *Infection* is also rare; but diphtheria has been seen, and we would recommend rather to postpone a tonsillotomy during an epidemic of diphtheria or scarlet fever. It does not seldom happen that the greyish-white fibrinous film, which is often deposited on the wound surface after operation, is thought to be diphtheric.

Evulsion.—Various authors, and recently *Winckler* also, advise evulsion of the tonsils (shelling out the tonsils), especially if the latter are fissured, deeply seated, and are often subject to inflammations. The arches are separated as much as possible from the tonsil, and then the tonsil is seized by a forceps and freed by means of an elevator without cutting. This done, the whole tonsil can be separated by rotating the forceps, aided slightly by the elevator. Children and nervous patients are anaesthetised (*Winckler*). Evulsion takes eight to twelve days to recover, but is said to have a good influence upon the voice.

The *after-treatment* consists in the application of ice, cold and liquid food, and gargles containing disinfectants. Gargles, however, should not be forced, for the formation of thrombi may be impeded by it.

VI. ACUTE AND CHRONIC INFECTIONS.

1. ACUTE EXANTHEMATA.

In **measles,** we very often see the mucous membrane of the hard and soft palate affected with a similar exanthem, which precedes that of the skin, and enables us, in conjunction with "*Koplik's* spots" (see p. 203), to make an early diagnosis and to isolate the patient in time, a circumstance which is very important in respect to prophylaxis.

Scarlatina.—The first (prodromal) stage of scarlet fever is dominated by angina. In doubtful cases an angina will always guide us towards scarlet fever. Measles mostly begins with coryza and conjunctivitis, seldom with angina.

In scarlet fever the angina shows two forms: (*a*) a benign form, in which the pathological changes are situated in the superficial layers, and (*b*) a malignant form, in which the morbid processes are situated in the deeper tissues. The former shows the signs and symptoms of an ordinary catarrhal or follicular tonsillitis; the latter leads to the formation of abscesses,—tonsillar, peritonsillar, and retropharyngeal,—to phlegmons in the neck, and even to gangrene of the mucous membrane. In all these cases the submaxillary glands of the neck are swollen and may suppurate.

From these forms of angina the so-called *scarlatinal diphtheria* must be distinguished. There can certainly be no doubt that diphtheria may be associated with one of the slighter or grave forms of scarlatinal angina. Why should *Klebs-Löffler's* bacilli not be admitted to the inflamed tissues of an anginous tonsil?

But that which is usually termed scarlatinal diphtheria, however, is not true diphtheria, but a disease caused by the establishment of streptococci, and resembles diphtheria, viz., pseudo-diphtheria, and is an angina leading to necrosis. Here, also, mild and grave varieties may be distinguished. Correspondingly, after four to five days, sometimes earlier, sometimes later, one sees whitish or whitish-yellow or greyish specks, or patches, or stripes, on the palatine arches, tonsils, or uvula, which may unite and form "false membranes"; and, in the very gravest form, called by *Heubner* "plague-like" forms, there is a brown or blackish coloured false membrane. In these latter cases the fibrinous exudation is not only on the surface, but extends also into the deeper tissues, and results in a "coagulation-necrosis" which is widely destructive of all the structures, and leads, at the termination of the disease, to extensive scars and adhesions, if not to death from septicaemia, or, in a rare event, from erosion of a large blood-vessel. The lymphatic glands of the neck are, of course, swollen, and there is often an unpleasant or even foul discharge from the nose.

Smallpox.—Apart from slight catarrhal symptoms, which are usually present, we find in smallpox an eruption on the pharyngeal mucous membrane, similar to the cutaneous exanthemata, which has been already mentioned. (See p. 203.) The dimple (umbilicated depression) in the middle of the pustule is characteristic of variola. The pustules are also frequently found in the nose and upper air-passages. There may also be coexistent a kind of diphtheric inflammation, which, like the pustules, may give rise to the formation of ulcers. Pain on swallowing and salivation are never absent.

Chicken-pox (Varicella).—Here the symptoms are much milder, and we usually see only redness and swelling (catarrh) of the mucous membrane, and, in rare cases, papules or vesicles.

Diagnosis.—In all these forms of angina the diagnosis should offer no difficulties if one takes into consideration the whole state of general disease, especially the simultaneous, or shortly following, outburst of a similar eruption on the skin, and the local signs, which are often (measles, scarlet fever, smallpox) characteristic. In scarlatinal angina, moreover, a microscopical examination will put the matter beyond doubt.

Treatment.—In slight symptoms local treatment is superfluous, or it is sufficient to clean and disinfect the mouth and throat as usual. This is, however, not to be neglected, more particularly in scarlatinal angina.

Here the drinking of acidulated lemonade is of great benefit to children, and older children could also use it as a gargle. Solutions of aluminium acetate (1 to 2 per cent.) or peroxide of hydrogen 3 per cent. may also be used as mouth-washes. *Catti* praises the atomising of sublimate solution 1:5000 three or four times daily; and in the case of small children, touching the respective parts with swabs of cotton-wool dipped in a concentrated solution of sublimate (1:1000). He has never had a case of poisoning or bad accident from it. *Heubner* orders cleaning with water, to be followed afterwards by gargles of ichthyol, and small children who cannot gargle are to be douched with ichthyol, the head being inclined forwards.

In serious cases of angina and pseudo-diphtheria *Heubner* advises submucous injections of 3 per cent. carbolic acid. Half a *Pravaz* syringe (0.5 c.c.), twice daily, is injected on each side (into the tonsil, arch, or soft palate) through *Taube's cannula* fixed on the syringe, until the urine shows the carboluric colour (brown or smoky). Ice applications around the neck may be useful in the beginning, but in cases of necrosis and suppuration ice is less appropriate. Enlarged lymphatic glands which do not quickly subside on the application of ice should be softened by hot fomentations and then incised.

Injections of serum (diphtheria antitoxin) are justified in those cases where *Löffler's* bacilli have been found.

2. TYPHOID FEVER (ENTERIC FEVER).

At the beginning of typhoid fever a catarrhal angina is not a rare occurrence. According to French authors, the mucous membrane shows sometimes here a rough, uneven, scaly appearance (angina pultacea), due to the peeling off of the epithelium. Superficial necrosis may occur in the region of the pharyngeal mucous membrane, particularly that of the palatine arches, where small round or oval, sharply defined ulcers are formed, with a greyish film covering the base. The patients complain of pain on swallowing. The presence of such ulcers, which, however, soon heal, should direct our attention to typhoid in all doubtful cases.

3. DIPHTHERIA.

Etiology and Pathology.—Pharyngeal diphtheria (diphtheria faucium; angina diphtherica) represents the most important, often the only, localisation of a specific disease caused by the *Klebs-Löffler* bacillus, and occurs mostly in children, seldom in adults. Infection takes place by contagion from person to person, the disease showing the characteristics of an epidemic or endemic. The germs also cling to inanimate materials (fomites), such as clothes, toys, food, and for a longer period on moist than on dry material. Of great importance is the circumstance that many persons themselves carry the bacilli about in their mouths, without being themselves affected by diphtheria. Such persons may transmit the bacilli to other more susceptible people, the transmitters themselves being immune. This is perhaps due to certain antibodies circulating in their blood, or they suffer only from a slight catarrhal or follicular angina. As a matter of fact, diphtheria may be disguised under the mask of such an angina. Thus it may be explained that persons having been in contact with patients suffering from diphtheria can transmit the disease to others who are more susceptible to it, without themselves being ill. This susceptibility is dependent on some certain, local or general disposition, viz., age, previous diseases (measles, whooping-cough, etc.), hyperplastic tonsils, adenoid growths, etc.

In the majority of cases, especially in the graver forms, a mixed infection has taken place; *i. e.*, besides the diphtheria bacilli, other microbes (streptococci) have established themselves in the body. It is, however, not yet ascertained how far the general disease in these cases is dependent on the action of the streptococci; and, moreover, such a mixed infection in the serious cases of diphtheria has been recently denied, and all the grave symptoms have been ascribed to the formation of toxins by the *Löffler's* bacilli (*Genersich, v. Ranke, Uffenheimer*). This toxin, which can be isolated and has been used for experiments by *Roux* and *Yersin,* is that which produces the grave general symptoms, and last, but not least, the paralysing action on the heart and nervous system.

Death from heart failure is thus the effect of the diphtheric toxin. The same toxin, not the bacillus, is also the cause of the diphtheric membranes in the throat, as *Roux* and *Yersin* have proved, and these same membranes also are able to block the air-passages (in descending diphtheria of the larynx) and cause death by asphyxia.

Fibrinous Pharyngitis.—A fibrinous exudation accompanied by fever occurs in the throat, especially on the tonsils, as well as in the nose, and forms false membranes, which can easily be separated, but after removal soon recurs. After the application of the galvano-cautery, and more especially of caustics, such fibrinous deposits may be seen. These deposits consist of fibrin, leucocytes, epithelial cells, and microbes. Though *Klebs-Löffler's* bacilli are said not to be present, one would act wisely in treating such a case of fibrinous angina as if it were diphtheria, no matter however slight it may be. The disease sometimes lasts several weeks.

Pathological Anatomy.—In true diphtheria fibrinous inflammation is combined with necrosis. If the process is situated in the *superficial* tissues, the vessels degenerate and a fibrinous exudation takes place under and upon the epithelium. Then the superficial epithelial strata undergo necrosis, to form the characteristic discoloured greyish-white pseudo-membranes, and which consist of a network of fibrin (fibrinous filaments), and between the meshes of which cellular elements may be found. These pseudo-membranes are firmly adherent to the underlying mucous membrane, and cannot be separated without causing some bleeding. If, on the other hand, the process occurs in the deeper tissues, a fibrinous exudation followed by necrosis takes place. Under the necrosed tissue the mucous membrane is inflamed and shows round-celled infiltration; the vessels are dilated, and, finally, suppuration sets in, and the pseudo-membrane, as well as the necrosed parts, is cast off and discharged.

Symptoms and Course.—The disease shows in the beginning, with regard to the general and local symptoms, the same picture as in simple angina. (See p. 263.) On inspection also it would be difficult to discover a difference. Only a careful survey or a bacteriological investigation would reveal to us the true nature of the disease, though not always. In pronounced cases inspection of the throat would probably not leave us in doubt. One sees on one or both tonsils smaller or larger sized greyish-white patches, which cover, as the case may be, more or less of the whole tonsil, and spread onto the adjacent parts, more especially onto the palatine arches, uvula, the lateral, and less frequently the posterior, pharyngeal wall. In certain cases the membranes are covered with discoloured muco-purulent fluid. The pseudo-membranes loosen after three or four days, and are then discharged, and are either spat out or coughed up or, as the case may be, swallowed. The defects caused by the detachment of the pseudo-membrane are repaired, or the pseudo-membrane is again renewed, until, finally, after two or three recrudescences, recovery is established.

In severe cases the process from the outset shows a tendency to extend in surface and depth. Here the loss of substance is much more extensive, and only heals with the formation of scars, owing to the mucous membrane itself being affected; afterwards adhesions may often be seen. The inflammation spreads upwards into the naso-pharyngeal space and nose (see p. 93), to the Eustachian tube and middle ear (in certain cases the disease may probably originate from the naso-pharynx), or it may invade the mouth or extend downwards into the larynx, trachea, and bronchi, and, with signs of severe dyspnoea, brassy cough, and hoarseness, lead to suffocation if not quickly interfered with. The progress and involvement of additional regions, as well as the renewed formation of pseudo-membranes in the throat, are on such occasion ushered in by an *increase of temperature.*

The most *severe* forms of diphtheria are characterised by signs of *gangrene and sepsis.*

In gangrenous diphtheria all the affected parts, and in particular the tonsils, are very much swollen. The diphtheric membranes appear of a greyish-green, dark-grey, or brownish colour if mixed or stained with the blood. The epithelium is separated, blebs occur, and extensive necrosis of all the tissues supervenes, making itself evident by a horrible foetor ex ore. If we examine the throat, we experience difficulty in recognising the configuration of the pharyngeal organs; the inspection is, moreover, impeded in many cases by haemorrhages from the various necrosed tissues. The lymphatic glands in the neck are, as a rule, enormously swollen.

The gangrenous form may originate from the pseudo-membranous form. The diphtheria must have already existed for several days, or even weeks, before the gangrene supervenes, and it is only in rare cases that the gangrene exists from the outset. The patients die rapidly with all the signs of sepsis, or an erosion of a large vessel in the neck may prove fatal. It may happen, however, that the gangrene is limited, and a line of demarcation is shown; thus recovery takes place after the separation and discharge of the foul and dead matters, leaving behind them scars or adhesions, such as are usually and only seen in severe syphilis.

The septic form of diphtheria is characterised from the beginning by the severeness and intensity of the general symptoms, high fever, small and feeble pulse, apathy, and drowsiness; whereas, on the other hand, the pain on swallowing is much less marked than usual. The lymphatic glands are likewise very much swollen, and ecchymoses of the skin and mucous membrane soon appear. The septic form originates either from, or is associated with, the gangrenous form, or it develops directly from the pseudo-membranous form, without the intercurrence of gangrene. The patients usually succumb in from two to three days, and death often ensues with a subnormal temperature and with the evidence of severe cerebral disturbance or by heart failure or oedema of the lungs.

Complications.—(1) *Nose, Mouth, Larynx.*—The diphtheric affection of the nose and mouth is usually an extension from that of the throat, and is seldom itself primary; and, as has been already stated, laryngeal diphtheria will be discussed later on in Part IV.

(2) *Heart.*—As has been already said, the heart is very often affected in diphtheria. The patients die of cardiac paralysis, and even also in seemingly mild cases, and perhaps during convalescence again, failure of the heart, with frequent and small pulse, might occur, and then death ensues.

If the pulse-rate increases to over 100 without a visible cause, affection of the heart is to be expected, and calls for the greatest precaution. In other cases the debility of the heart is more gradual; the children become more and more apathetic or somnolent, and finally die from exhaustion. In the rapid cases of cardiac failure we must attribute it to the effect of the toxins on the vagus and cardiac ganglia, and in the more gradual cases we usually find degeneration of the myocardium and endocardium.

(3) *Kidneys.*—A slight cloud in the urine is always present as the result of the fever. In the more serious cases a large amount of albumin in the urine indicates a parenchymatous nephritis.

(4) *Skin.*—It sometimes happens that by inoculation of the virus into cutaneous fissures or excoriations the skin becomes affected, and the disease is apt to extend superficially, as well as into the deeper tissues.

(5) *Eye.*—Diphtheric conjunctivitis is not often observed; and for the symptoms and treatment see text-book on Diseases of the Eye.

Sequelæ.—With regard to the matter of after-affections, we are mostly called upon to deal with paralyses, which are probably the result of peripheral neuritis, due to the diphtheric toxins. Post-diphtheric paralyses usually occur about the beginning of the third week of the disease, but sometimes still later.

Paralysis of the soft palate is the most common, and manifests itself by the regurgitation of food through the nose, and by rhinolalia aperta, the velum palatinum being immobile during intonation or speaking, and shows also marked anaesthesia.

Very common, likewise, though perhaps less frequent than the foregoing, is *the paralysis of accommodation*, causing inability to read or see closely (paralysis of the ciliary body). Other ocular muscles may be paralysed, especially the external rectus. *Paralyses of the vocal cords* have been observed—unilateral as well as bilateral. If the *sensory branches of the laryngeal nerve* (superior laryngeal nerve) are paralysed, the danger is great, for the patients, owing to the abolished reflex action, are prone to misswallow (dysphagia), and become subject to "foreign body pneumonia." *Paralyses of the skeletal muscles or limbs* are least often observed, as also is ataxia, viz., a disturbance of coördinated movements. The patients are not paralysed, but their movements are ataxic, clumsy, and unskilled. The *knee-jerks* are often abolished, at least for a certain time. Many of these motor and sensory lesions may be combined.

Paralysis of the myocardium, which may also occur during the convalescent stage, has already been mentioned. *Paralysis of the muscles of respiration*, which is luckily very rare, causes death by asphyxia.

All these nervous lesions in general mostly occur in severe forms, but may also appear in the milder forms.

Diagnosis.—Every experienced practitioner will agree that there are cases which are difficult to diagnose, and can be elucidated only by microscopical examination, viz., finding of *Löffler's* bacilli. Many cases of follicular or lacunar angina (see p. 264) may be mistaken for diphtheria, or slight cases of diphtheria mistaken for follicular angina. If a bacteriological examination, whether by culture in a test-tube or on a specially prepared nutrient plate, or by the microscope alone, is not readily obtainable or is impossible, then it is as well to treat each doubtful case, especially in times of epidemics, as if it were really diphtheria, and isolate it. The pathognomonic membranes, which cannot be separated without some force and without bleeding, taken in combination with the whole general picture of the disease, will admit of a diagnosis. Severe cases, in an advanced stage, or if followed by complications, are not easily mistaken. As to fibrinous pharyngitis, see p. 301.

Prognosis.—Generally speaking, the prognosis is less favourable in children than in adults, and it is still more so the younger they are. On the whole, it depends much on the character of the epidemic, on the "genus epidemicus," which itself again depends on the virulence of the bacilli. There are slight cases, healing in from one to two weeks, and more serious ones, which require three or four weeks and more for recovery. If the process shows a tendency to spread and the membranes are found on other regions as well as on the tonsils, the prognosis becomes graver; and it is bad if the process extends to the deeper air-passages (larynx or trachea). The septic and gangrenous processes likewise render the prognosis bad, and it is still worse if the glands are much swollen, if the fever is high or subnormal, and if the pulse-rate is subject to great oscillations. a circumstance the importance of which cannot be overrated. A feeble and frequent pulse, but also a slow and irregular pulse, always points to danger from cardiac failure. Patients whose hearts have already been weakened by a previous infectious disease are, in my opinion, worse off should diphtheria attack them.

The prognosis has been improved as the result of the introduction of serum-therapy, at least in the opinion of the majority of physicians, provided that the serum is applied at an early stage of the disease and in sufficient strength and quantity.

Diphtheric paralysis usually affords a good prognosis. It is only the paralyses of the respiratory, phrenic, and laryngeal muscles that are dangerous.

Treatment.—(*a*) *Serum-therapy.*—The introduction of the anti-diphtheria serum makes it our duty to inject it in each case of assured diphtheria. It cannot be denied that many cases would have recovered even without the serum; and it is just these mild cases in which the usefulness of the serum has been doubted; but one should never forget that diphtheria is a disease which is incalculable in its course, and that the serum is the less valuable the later it is applied, and the longer the body has suffered from the deleterious influence of the diphtheric infection (as to the method of application, see p. 96, et seq.).

We here desire to say only a few words on the dosage. In the milder cases, and in children under three years of age, 500 units of antitoxin are sufficient.

In older children, up to six years, 600 to 1000 units are required. In patients over six and in advanced cases 1500 units should be injected at once. Severe cases require the immediate injection of 2000 to 3000 units. The injection must be repeated, and on repetition a smaller dose is required, and, as the case may be, this may be yet once more repeated. The effect of the injection is often astonishing. The local process becomes limited, the pseudo-membranes are cast off, the fever abates, and after twenty-four hours convalescence is well established.

(*b*) *Local Treatment.*—The use of antitoxin mostly renders every other form of treatment superfluous, but if it is found necessary, disinfectant gargles or mouth-washes may be ordered. Little children's throats can be sprayed or douched with lime-water or aluminium acetate (one teaspoonful in a tumbler of water); the head being preclined, this certainly renders feeding easier. For older children and adults I am accustomed to prescribe formamint tablets. Ice applications around the neck are very useful in severe cases.

In any case *careful attention must be paid to the heart.* In threatening syncope stimulants are required, such as wine, caffein, camphor (½ to 1 syringe of camphor oil). Above all, the heart must be strengthened by a strongly nourishing diet (milk, meat-juice, wine with egg-yolk, etc.), and children *must be kept quiet in bed as long as possible.*

Complications and sequelæ must be treated according to the requirements. In nephritis, a suitable diet is most important (milk, Wildungen spring, etc.), and tepid, and later hot, baths are very useful. Paralyses are treated by electricity. (For the treatment of laryngeal diphtheria, see Part IV.) With regard to prophylaxis, strict isolation of the patient is necessary, and disinfection of the sick-room indispensable, and in this respect also all the surroundings (toys, linen, books, etc.) which have been used by the patient must be disinfected. Children belonging to the same family attending school ought to be kept away.

The question as to the preventive value of *Behring's* serum is not yet settled; anyhow, a prophylactic inoculation can be recommended, although the immunity derived therefrom lasts only from one to two weeks. For the purpose of immunizing children, 200 to 300 units of antitoxin serum are required.

4. TUBERCULOSIS AND LUPUS.

Etiology.—Lesions of the epithelium or catarrhal changes of the mucous membrane of the pharynx and naso-pharyngeal space, and especially of the tonsils, in children, are much favoured by the germs of tuberculosis and lupus.* These affections are usually of secondary nature, as in the case of tuberculosis following that of the lungs or larynx, and the lupoid affections usually spread from the nose or external skin into the pharynx. The throat, however, may also be the primary seat of tuberculosis, although it might be difficult in a given case to discover any other tuberculous lesion. This disease occurs at any age. Lupus seems to occur more frequently in young persons, while children are comparatively immune.

Pathological Anatomy.—The reader is referred to the same disease of the nose. (See p. 97.)

Symptoms and Course.—(1) *Tuberculosis* is mostly found on the velum palatinum, on its arches, and on the uvula; it sometimes occurs on the posterior pharyngeal wall and lateral folds; seldom, apparently, on the tonsils, though these organs, as has been said, form the entrance gate and nidus for the bacilli. In the pharyngeal tonsil the tubercular process is often latent; neither does it show any marked symptom, even if seated in the palatine tonsils. (See Fig. 85.)

** Grober* injected fine Chinese ink into the tonsils of rabbits, and showed the connection between the tonsils and the lymphatic system of the neck, chest, and pleurae.

If an opportunity occurs to observe a case in its early stage, on inspection, the mucous membrane is seen to be reddened and infiltrated by a gelatinous fluid, and scattered among which are the tubercles, shining through like poppy or millet seeds, as greyish or yellowish nodules.

Later on, these nodules can be observed breaking down to form small flat ulcers with irregular notched edges. On the margin of these "lenticular ulcers," or in their vicinity, other nodules or ulcers may be noticed. The ulcers become confluent, and unite to form larger-sized ulcers or infiltrates. The ulcers appear atonic, *i. e.*, show little inflammation, and have a tendency to spread superficially, therefore, the formation of a tuberculoma is very rare.

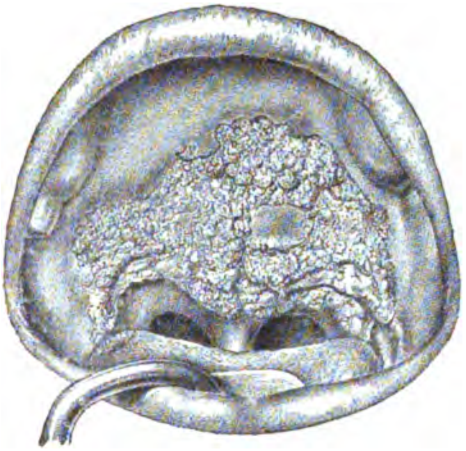

Fig. 85.—Tuberculosis of soft palate (from the Vienna laryngological clinic).

As a rule, the patient comes to the physician when a more or less extensive ulceration has taken place. Accordingly, pain radiating to the ear is very troublesome, and various forms of previous paraesthesias are mostly disregarded and not mentioned. The pain becomes more severe in proportion to the spreading of ulceration, and is apt to hinder feeding. The free motion of the soft palate is impeded, the voice becomes nasal, and fluid regurgitates through the nose. The lymphatic glands in the neck are swollen, and foetor ex ore may exist. The ulceration spreads along the base of the tongue, and is liable to extend from there onto the lateral walls of the pharynx and larynx. The general health suffers much, the patient soon becomes run down, especially on account of deficient nourishment and fever, and other tubercular diseases (lungs), as well as the agonising pains, soon exhaust the patient.

(2) *Lupus* follows a milder course. Small nodules, which look almost like granulations, clustered and in great numbers, may be seen. Any portion which breaks down tends to form shallow little ulcers, which have the same atonic appearance as do ordinary tubercular ulcers, but can be distinguished from them by the great number of newly formed nodules on their margins. In long-standing cases the formation of scar tissue, which has a great tendency to contract, and is situated between the nodules and ulcers, is conspicuous.

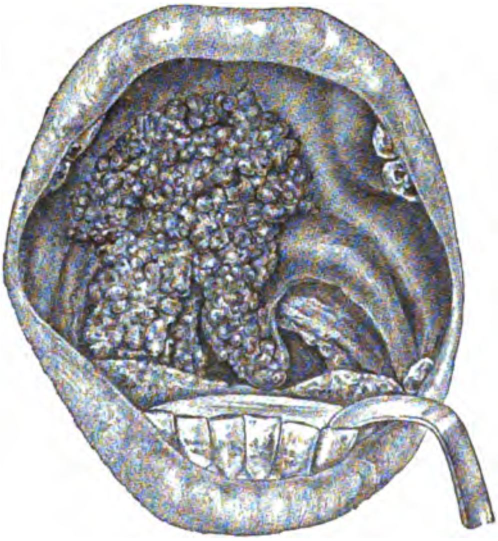

Fig. 86.—Lupus of the soft palate (*Türck*).

The subjective symptoms are much less pronounced in lupus than in tuberculosis, and the progress of the disease is also much slower. (See Fig. 86.)

Diagnosis.—In some cases the decision, whether we have to deal with tuberculosis or lupus, or if there is any other ulcerative (syphilitic) process present, especially if the examination of the body is negative, is not easy. *Tubercular and lupoid ulcerations do not spread in depth,* as do the syphilitic lesions, and have always a more or less atonic appearance, and are characterised by *the nodules on the margins.* In doubtful cases microscopical examination and iodide of potassium will aid the diagnosis. If the external skin shows no lupus, distinction between tuberculosis and lupus might be impossible. *Formation of scars inclines opinion in favour of it being lupus.*

Prognosis.—Pharyngeal tuberculosis, on the whole, possesses an unfavourable prognosis, although if primary, recovery is not excluded; but mostly the decreasing powers, the progressive inanition, and tubercular processes in other organs soon lead to dissolution. The discovery of giant-cells or tubercle bacilli in an excised pharyngeal tonsil ought not to have any influence on our judgment, for a latent tuberculosis of the pharyngeal tonsil does not seem to be a source of infection for other organs. Lupus, perhaps, has a better prognosis, but is inclined to relapse.

Treatment.—If the disease is not yet too far advanced, the ulcerations may be thoroughly scraped away by means of the sharp scoop, and afterwards cauterised with lactic acid (30 to 80 per cent.), provided that the patient's state of health admits of such rough handling. Otherwise we must treat symptomatically; *e. g.*, relieve pain by painting with cocaine (5 to 10 per cent.), or menthol (20 per cent.), or insufflation of equal parts of orthoform and iodol. *Holländer* recommends iodide of soda twice daily internally and the insufflation of calomel. This is said by *Holländer*, who does not make a principal difference between lupus and tuberculosis, to have a selective caustic effect and, at the same time, to saturate the tissues with iodine, which is supposed to have a specific effect upon tubercular processes. General measures of treatment are of the greatest importance.

5. SYPHILIS.

The pharynx is so often subject to syphilitic disease that one should never omit the examination of the pharynx of a person who is afflicted with or is suspected of having syphilis. There are only a few cases in which the pharynx shows no manifestations of secondary syphilis; where these seem to be absent, they are probably overlooked, owing to their causing very little discomfort. The late secondary stages or tertiary symptoms are not so frequent, yet are common enough; on the other hand, however, the primary lesion is seldom found localised on the pharyngeal mucous membrane.

(*a*) *Primary Lesion* (*Initial Sore; Hard Chancre*).—The usual seat of the initial affection is the tonsil. It here forms a superficial erosion or ulcer, the edges of which are indurated and rolled over like a brim; its floor is covered with a smeary greyish film, and it usually causes no trouble, though in some few cases pain on swallowing is very considerable. The glands on the same side of the neck as the chancre are always swollen and indolent. (For the chancre of the tubar ostium see p. 99.)

(*b*) The *secondary signs* are analogous to those in the like condition of the mouth (see p. 207).

The erythematous (macular) syphilid occurs as part of the picture of *syphilitic angina*. The soft palate and tonsils are diffusely reddened, *contrasting sharply* with the paler, healthy tissue, and they cause little or no pain. The specific nature of the angina could not be recognised if it were not for the connection with symptoms and signs elsewhere on the body (swelling of the lymphatic glands, painlessness, and other syphilids). Syphilitic angina is marked by its obstinacy.

The papular form is represented by the "plaques muqueuses," which in appearance exactly resemble those of the oral mucous membrane.

They occur most commonly on the palatine arches, uvula, tonsils, and less commonly on the lingual tonsils, and sometimes on the posterior pharyngeal wall. The oral mucous membrane also is nearly always implicated, but it does not rarely occur that only a part of the anterior palatine arch or tonsil is affected, showing irregular yellowish-white patches, not dissimilar from pseudo-membranes. Such cases sometimes offer difficulties in diagnosis. The condylomata having subsided, the tonsil shows a tuberous, irregular surface, especially if there was ulceration from the breaking down of the papules.

(*c*) *Tertiary symptoms* are also usually associated with such as are in the mouth. In the naso-pharyngeal space isolated gummata sometimes occur, especially on the posterior surface of the velum palati. If we only have the slightest suspicion of syphilis, in cases of pain on swallowing, radiating to the ear, together with a paresis of the soft palate, it is our duty to examine by posterior rhinoscopy, and should this prove impossible, to ascertain ex juvantibus the specific nature of the disease by large doses of iodide of potassium.

If the above is not done, one may wait, only to see the softening of the gumma, which will lead to perforation of the soft palate and the extensive defects thereof. (See Fig. 87.)

Comparatively little discomfort is caused by *a gumma of the hard palate. The posterior pharyngeal wall* is sometimes also the seat of a gumma, without giving any trouble; and it is then the ulceration itself which leads to a correct diagnosis, or one may be only able to see the ulcer alone, with its crater-shaped and indurated edge and filmed floor. It is necessary, in some cases, to lift the velum with a probe in order to see more clearly the ulcer, which usually has a tendency to spread upwards.

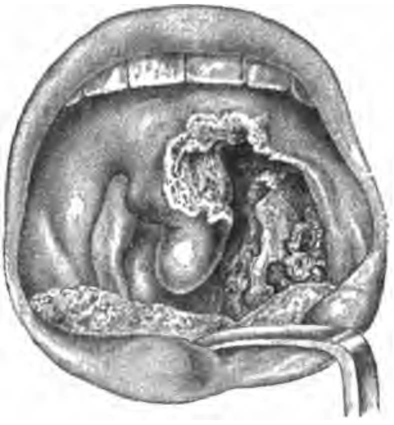

Fig. 87.—Gummatous ulceration of the soft palate in left tonsil, and the posterior pharyngeal wall (*Türck*).

The scars remaining after the ulceration has cleared up are white, glossy, and radiating, and cause more or less considerable deformities by their contractions, or are followed by adhesions or obstruction if they form fibrous bands and connections between the surfaces concerned.

Diagnosis.—Hard chancre of the tonsil may be mistaken for carcinoma, gummatous ulcer, or necrosing diphtheria. With regard to the differential diagnosis between gummatous and carcinomatous ulcer and chancre the same holds good, mutatis mutandis, as was said on page 210. Diphtheria is mostly bilateral and presents a more rapid course.

With regard to the *secondary manifestations*, we may refer to what has been said previously.

Plaques muqueuses, if localised on the tonsils, are difficult to discriminate from other affections, and are often not recognised; and it is just the same with syphilitic angina. As in other localisations of syphilis, the diagnosis is, in many cases, only made after longer observation or from the effects of mercurials or iodides.

The difficulties in the diagnosis of the tertiary symptoms have been discussed in Diseases of the Mouth and Nose, to which we refer. There is now hardly anything to be added. The importance of posterior rhinoscopy has been pointed out. In general, syphilis should be always our first thought if we have to deal with ulceration in the pharynx existing for a more or less prolonged period, especially if causing no trouble, nor pain, nor fever. A certain polymorphism, as to change and alteration of the appearances, and also as to size and seat, is characteristic of syphilis.

Prognosis and treatment have been already discussed in various chapters. The treatment of post-syphilitic adhesions and deformities has been treated in the chapters on Malformations and Deformities.

6. SCLEROMA (PHARYNGOSCLEROMA).

Pharyngoscleroma nearly always occurs in association with rhinoscleroma, and is either a continuation of the nasal process or is a primary affection of the naso-pharyngeal space, spreading into the nose and down the throat.

Etiology and pathological anatomy have been discussed in the chapter on Rhinoscleroma. Here it remains only to speak of the changes with respect to the special anatomical conditions of the pharynx.

One finds here also, as in the nose, nodules of well-defined or more diffuse infiltrations, the former being more frequent than the latter. These infiltrations are found in various places, most frequently on the lateral pharyngeal wall, velum palati, and its arches. Later, the infiltrations undergo atrophy and shrink, with the formation of scars and fibrous bands, which may lead to gross distortions, adhesions, and deformities of the respective parts. The soft palate is drawn backwards, so that the view of the naso-pharyngeal space is blocked, and the uvula may be found pulled right up on to the soft palate. If the process spreads to the anterior palatine arches, the tongue becomes involved and fixed, and if the cheek is implicated, fixity of the jaw may result from the fibrous ankylosis.

The discomforts are directly dependent on the size and extent of the infiltrations and shrinkages. Breathing through the nose may be impeded or entirely obstructed, and speaking, chewing, and swallowing may be considerably inhibited; hearing also may be impaired, and there are often buzzing noises in the ear.

Diagnosis.—The diagnosis would hardly offer difficulties in pronounced cases, though it might be confounded with tertiary or gummatous infiltrations. But the course of the gumma is always much quicker than that of scleroma, and, besides, gummatous infiltrations never show the hardness and resistance that scleromatous ones do. In doubtful cases a trial with iodid of potassium and a microscopical examination, viz., the finding of scleroma bacilli, will be decisive. The distinction between syphilitic and scleromatous scars is more difficult. Generally speaking, syphilitic defects are usually greater and more extensive than those produced by scleroma.

Prognosis.—The prognosis is identical with that of rhinoscleroma.

Treatment is also the same, and is principally symptomatic and of little avail. (We refer to the chapter on Rhinoscleroma, see p. 105.)

7. MALLEUS (GLANDERS).

Manifestations of malleus in the throat have no *features distinct* from the nasal disease of the same name, which follows on or occurs at the same time with it. There are small ulcers on the posterior pharyngeal wall, and, less often, nodular or diffuse infiltrations. For the rest, we refer to what we have said on malleus of the mouth and nose (see p. 107).

8. LEPROSY.

According to *Bergengrün*, *tuberous* leprosy never, and *anaesthetic* leprosy seldom, occurs on the pharyngeal mucous membrane. The pharynx and naso-pharyngeal space are usually affected before the larynx, but later than the nose. The seat of predilection is the soft palate and uvula. At first catarrhal symptoms are prevalent, but later on infiltrations occur and are more or less hard, knotty, or nodular, and which are perfectly anaesthetic and tend to break down. In very marked cases the pharynx has a certain resemblance to a stalactitic grotto. The mucous membrane, which is at first moist and shiny, later on becomes very dry, and the knots and nodules break down, and so result in extensive destruction, and finally the formation of scars follows the ulceration.

Diagnosis.—The diagnosis should offer no difficulties if we take into consideration the whole picture and other signs and symptoms.

Treatment is symptomatic (see p. 108).

VII. MYCOSES.

1. THRUSH (German, Soor).

The thrush fungus, the pathology of which we have already described in a previous part (p. 212), is occasionally found in the pharynx, whence it spreads from the mouth and sometimes causes pain on swallowing.

Diagnosis.—The differential diagnosis must be made from diphtheria; the latter, however, shows a much more rapid course. The pseudo-membranes of diphtheria cannot be separated without causing bleeding, while the tufts of thrush can be wiped off, although some force has to be used without producing bleeding. The diagnosis will be assured by a microscopical examination.

Prognosis and Treatment.—As to prognosis and treatment see chapter on Thrush in Mouth, p. 212, et seq.

2. LEPTOTHRIX (HYPERKERATOSIS PHARYNGIS; LEPTOTHRIX PHARYNGO-MYCOSIS).

Etiology and Pathology.—As in an analogous disease of the mouth, so here, it consists of the invasion of the leptothrix fungi, which, together with the corneous epithelial cells, form spinous excrescences and soft or hard concretions or cones, which project from the tonsillar lacunae, but may also be found at the base of the tongue, in the region of the lingual tonsil, or on the lateral or posterior pharyngeal wall. The lateral folds sometimes appear as if strewn with them. The mucous membrane in the vicinity shows a normal appearance or is slightly injected.

Symptoms.—There are few complaints on the part of the patient. Sometimes a nervous patient is upset at having discovered something white in his throat, or he may feel a sensation of dryness, or tickling or burning, or have the sensation of the presence of a foreign body in the throat.

On examination, one finds the concretions and cones, which must not, however, be confounded with those arising from inflammatory processes in the tonsils. These are firmly adherent, difficult to remove, and are soon renewed.

Diagnosis.—Pharyngo-mycosis may be distinguished from diphtheria, as it is afebrile, slow in its progress, and causes no discomfort. The mycotic cones or concretions do not change their form nor unite. The differential diagnosis between mycosis and tonsillar concretions is more difficult. The latter, however, are soft and are localised to the region of the tonsils, which, moreover, show signs of previous chronic inflammation.

Prognosis is characterised by the obdurate course and disposition to relapse. In some cases, however, the disease spontaneously disappears.

Treatment is powerless, and, therefore, superfluous where no troubles are complained of. *B. Fränkel,* who first described the disease, recommends painting with pure alcohol. *Rosenberg* maintains that women are more subject to it than men, which would seem to show that tobacco plays a certain rôle. Indeed, smoking has been advised as a remedy. This looks like exorcising the devil by the aid of Beelzebub!

3. OTHER MYCOSES.

Under this heading may be mentioned *pharyngomycosis sarcinica,* due to the sarcina fungus, and *pharyngomycosis aspergillina,* caused by the various kinds of aspergillus. Sarcina is found comparatively often in the throat and mouth. Aspergillus grows primarily mostly in the nose, and is of very rare occurrence. It forms white or grey patches, but causes little or no trouble.

Treatment, if of any value, consists in gargling or painting with disinfectants.

VIII. FOREIGN BODIES.

Etiology.—Foreign bodies reach the throat by way of the mouth. They consist usually of pieces of food, as, for instance, a morsel becoming impacted in the throat, or indigestible and hard substances, such as pieces of bone, fish-bones, kernels, fruit-stones, or splinters of wood, paper, etc., which perchance have been mixed with the food; or they comprise other things, such as needles, pins, and nails, which by a wide-spread though bad habit are commonly held between the teeth; bristles from tooth-brushes, stalks, corn-husks, coins, buttons, stones, artificial teeth, etc. Insects may occasionally also fly into the throat. Sometimes particles of food are thrown into the naso-pharyngeal space during the act of vomiting or in paralysis of the soft palate; they are usually flung into the nose. Again, it seldom happens that foreign bodies reach the throat by way of the nose; and still yet more seldom, from the ear or through the soft parts, in a case of injury.

Symptoms and Pathology.—Pointed or sharp-edged foreign bodies are often lodged in the tonsils or root of the tongue, but also in the valleculae and deeper portions of the pharynx, the entrance to the larynx, oesophagus, and pyriform sinuses. Larger foreign bodies are prone to slip downwards, and under certain circumstances are wedged fast, in such a way that they are liable to cause death by suffocation, by sudden compression of the larynx or the aditus ad laryngem. Very sharp-pointed foreign bodies, such as fish-bones and bristles, are sometimes so deeply buried in the tonsil or root of the tongue that they disappear entirely within it, or project only a very little way from the surface, and in other cases they are lodged transversely across the pharynx like fine threads of mucus.

Patients complain of pain on swallowing; of a sensation of pricking, which, however, cannot be always exactly localised. The power of localisation is in general very unsatisfactory. The patients, who are usually very much excited, often report that they feel the pricking or the pain caused by the foreign body in this or that region, though no foreign body can be discovered there or in the vicinity. It even happens that the foreign body has already been ejected and the patient still feels it or imagines that he feels it, whereas he can only feel the pain caused by the injury to the respective parts. After a few days these sensations have usually passed away. Infective foreign bodies, or such as remain *in situ* for a long period, are apt to excite phlegmonous inflammation, or the patients may scratch or scrape with their nails in the throat until they have injured and infected themselves. Occasionally serious haemorrhage may occur through injury or erosion of a large blood-vessel.

Diagnosis.—If the patient is not too excited and is sensible, he can be examined without difficulty. The parts which are mostly concerned must be thoroughly inspected under a good illumination, *i. e.*, tonsils, root of the tongue, valleculae, and the sinus pyriformis. A laryngeal mirror will be found useful. Larger foreign bodies will be easily discovered. Difficulties may, however, arise during the search for bristles, fish-bones, needles, etc. If they cannot be seen, the parts should be thoroughly *palpated.* If, nevertheless, nothing can be discovered, we might calculate on the possibility of the foreign body having been already thrown out and that the pain complained of is that due to the slight injury of the mucous membrane. But, we should also bear in mind that a man may have swallowed two fish-bones at once, or that one portion of the foreign body may be removed, and yet the other part still remain behind.

Any examination is difficult in overexcited persons, or is made difficult by the choking on the part of the patient. This often happens in patients whose mucous membrane has become hypersensitive, through the repeated and futile endeavours at extraction. In a case such as this, a firm, quiet attitude must be preserved and cocaine applied. In urgent cases the *x*-rays will be found very useful, though they will not reveal every kind of foreign body.

Prognosis.—Is mostly favourable. The subjective and objective symptoms usually disappear with the foreign body, provided that no infection or greater lesion has taken place.

Treatment.—If a foreign body is discovered, it should be removed at once, and for this purpose a suitably bent tweezer or forceps may be used, or the foreign body, if lodged in the upper part of the pharynx, can be extracted with the finger. If a rounded object sticks in the pyriform sinus, it must be loosened and brought into a suitable position for extraction. But great care should be taken not to allow it to pass into the larynx. Never try to push pointed or sharp bodies into the oesophagus, in order to avoid dangerous injuries. In cases of threatened suffocation we must perform tracheotomy if attempts at extraction of the foreign body do not quickly succeed, and in some cases pharyngotomy will be required.

APPENDIX.

Calculi.—*Etiology.*—Calculi of the tonsils resemble the concretions already mentioned, and are the products of chronic inflammation. They are caused by the drying up of the secretion and the deposition of lime salts in the lacunae.

Pathological Anatomy.—The stones or calculi consist chiefly of carbonate and phosphate of lime and organic matter. They may attain the size of a hazel-nut, are either smooth or spiky, and of a white, grey, green, or brownish colour. They act like a foreign body, *i. e.*, as an irritant, and may cause, or are associated with, abscess of the tonsil.

Symptoms.—The chief troubles are derived from inflammation or suppuration of the tonsil. The patient complains of pain on swallowing, which may also exist without inflammation, and of a peculiar sensation of tension in the side of the neck.

Diagnosis is easily made by palpation and probing. It might occur, however, that the tonsil shows ossification in various places, owing to ossifying metamorphosis of the fibrous tissue, or are due to ossified vestiges of the foetal branchii, or that an abnormally long styloid process has perforated the tonsil. (See p. 255.) In the latter case the bony process can be traced upwards and laterally, above the tonsil.

Treatment must be operative, and the calculi must be removed.

IX. TUMOURS.

1. BENIGN GROWTHS.

Some of the so-called benign tumours are congenital. To this class belong the teratomata, and hairy epidermoid polypi of the posterior pharyngeal wall and velum palati.

Other non-malignant growths are the *papilloma,* fibroma, angioma, lipoma, and enchondroma, the *fibroma* being the most common of them all. The fibroma is found, single or multiple, in the oral part of the pharynx, viz., on the uvula and palatine arches, where it forms round, smooth, or cauliflower-like growths from the size of a pin's-head to that of a pea. They may be pedunculated or sessile, and in the latter case are immovable on touching with the probe.

The papilloma originates less frequently in the tonsil. Here it consists of a short, stalked fibrous growth, showing the structure of the tonsils, *i. e.*, lymphadenoid tissue (*adenoid polypi*). These represent the same tumours, which we already know by the name of *tonsilla pendula.* (See p. 255.) *Cavernous angiomata* are seen on the uvula and palatine arches, but also on the posterior and lateral pharyngeal wall. They form dusky or bluish-black tumours, which sometimes attain considerable size. *Cystic formations* are occasionally seen on the roof of the naso-pharyngeal space, where the retention of secretion due to the blocking of a tonsillar recess (*bursa pharyngea*) may give rise to the formation of a *retention cyst.* (See p. 276.)

We must especially mention a certain class of tumour encapsulated between the two laminae of the palate, and known as *intramural tumours.* Their structure is that of a fibroma, myxoma, lipoma, or chondroma, and these are originally derived from the endothelium of the lymph- or blood-vessels. They are said to sometimes assume a malignant (sarcomatous) character. They are seated mostly on the hard or at the line of junction between hard and soft palate, are of a globular shape, are more or less soft in consistency, and can be distinctly moved under the intact mucous membrane, which is very often thinned out over them. In rare cases the thinned-out mucous membrane gives way and ulcerates, and in such a state conspicuously resembles a gummatous growth.

Symptoms.—The symptoms caused by these tumours depend on the size and seat of the growth, and very much upon the irritability of the patient. Small tumours on the soft palate or tonsils cause no discomfort and are usually discovered perchance; if the tumour, however, is larger and pendulous, it causes coughing or choking, by touching the root of the tongue or epiglottis; the patient has then a sensation of tickling and as of a foreign body; he has frequently to retch, and the voice might alter and even the respiration be obstructed.

Diagnosis might only prove difficult in the case of an intramural growth. Here the *confusion with a gumma* is very pardonable. However, gummata are mostly multiple, are not, or very little, movable, and show marked reaction with potassium iodide. *Malignant tumours* grow rapidly, break down early, and are mostly accompanied by swelling of regional glands, which rarely occurs in the case of benign intramural endotheliomata. *Aneurysms* occur very seldom in this region, and, moreover, would show pulsation. If they occur, they are found on the posterior pharyngeal wall, and are perhaps not true aneurysms, but are probably only abnormally large-sized branches of the ascending pharyngeal artery, which, under normal conditions, lies immediately under the mucous membrane, where it can be felt pulsating.

Prognosis is good.

Treatment.—Angiomata causing no trouble are best left alone; otherwise they must be removed by scissors or snare. Broadly seated tumours must be scraped away or destroyed by the galvano-cautery. Intramural tumours only require the covering mucous membrane to be incised, and are then easily shelled out.

The typical naso-pharyngeal polypi and retropharyngeal goitre are usually classified with benign tumours. With respect to their structure this is justified, the more so as they never produce metastases in other organs. But a certain malignancy cannot be denied to them, because of their incessant growth, whereby important organs are endangered, and of their changing their character by sarcomatous or cancerous degeneration. For this reason we will reserve for them a special description.

(a) Typical Naso-pharyngeal Polypi (Naso-pharyngeal Fibroma).—*Etiology and Pathology.*—Naso-pharyngeal polypi usually arise from the fibrocartilaginous *basilaris* (basal cartilage of the occipital bone), at the roof of the naso-pharyngeal space, and seldom spring from the vicinity. They are almost exclusively met with in males at the time of puberty, a state of affairs which is perhaps connected with some conditions of development of the skull, and is peculiar to men. After the twenty-fifth year they often undergo spontaneous atrophy.

Pathological Anatomy.—In uncomplicated cases the naso-pharyngeal polypus is a round, globular, rather hard, but nevertheless elastic, tumour, of yellowish-white or pinkish colour, with a smooth or slightly uneven surface. Usually single, it fills the naso-pharyngeal space, and because of its incessant and rapid growth, displaces the neighbouring parts and organs. It soon softens on the surface, giving rise to ulceration and adhesions, and invades the skull by sending out processes along preformed or self-created paths, as it grows.

Histologically, it consists of dense fibrous tissue and more or less agglomerated cells. Its blood-vessels are very numerous (angiofibroma).

Symptoms and Course.—The patients are usually first seen when the growth has already been in existence for a long period. In the forefront of the symptoms stands *obstruction of the nose* with its consequences: rhinolalia clausa, mouth-breathing, snoring, dryness in the throat, discharge from the nose, etc. Through occlusion of the Eustachian tube hearing is impaired and noises arise in the ear. If not interfered with, the tumour goes on growing, pushes forward the soft palate, invades the neighbouring cavities, leading to disfigurement and expansion of the nose and face, and especially in the regoin of the temple and cheek; causes exophthalmus, and, finally, leads to severe cerebral symptoms, such as intracranial pressure, giddiness, vomiting, somnolence, optic neuritis, disturbances of vision, etc. If the polypus grows downwards, dysphagia and dyspnoea, due to the blocking of the aditus laryngis, are the consequences. It may be noted that haemorrhages of a threatening and severe character are frequent. Owing to oft-repeated haemorrhages, the dysphagia, and disturbed sleep, the patients become very weak, so that they may die from exhaustion, if not from an intercurrent meningitis, or an attack of dyspnoea finishes him. In some cases the tumour becomes sarcomatous; in others, it is the subject of spontaneous retrogressive metamorphosis.

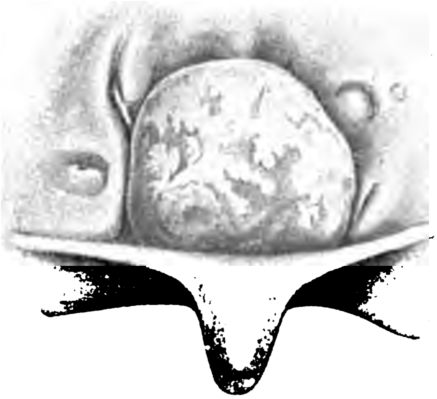

Fig. 88.—Typical naso-pharyngeal polypus (*Mikulicz*).

Diagnosis.—In advanced cases, where the obstruction of the nose is marked, and the voice has become thick and the face disfigured, the diagnosis will not long be doubtful. One examines the nose and finds it filled with a red tumour, which is covered by a mucoid secretion, shows perhaps ulceration, and proves to be resistant but elastic to the touch with a probe. If the slightest force were used while sounding, unpleasant haemorrhage might cause surprise. On inspection of the pharynx the velum palati will be found pushed or bulged forwards and paretic. Or perhaps the tumour is seen depending from the roof down into the oral section, or, if still comparatively small, is discovered by posterior rhinoscopy. (See Fig. 88.)

In unique cases one finds only one single red mass completely filling the naso-pharynx, covered by the smooth mucous membrane, or showing ulceration at various places, and coated with viscous secretion; but we here also observe the tense elastic quality. It may be difficult to ascertain where the tumour is attached, and how far it has undergone adhesions to the neighbouring or adjacent tissues.

It is also very difficult to distinguish between ordinary naso-pharyngeal polypus and sarcoma, for histologically they are both very much alike. We admit that many naso-pharyngeal polypi may be regarded as sarcoma.

Prognosis.—On account of their disposition to continue growing incessantly and rapidly, and their inclination to sarcomatous degeneration and recurrence, the prognosis cannot, from what has been said, be called a good one, though there is always the chance of spontaneous involution.

Treatment.—There are two modi operandi: (*a*) *Conservative,* by attacking the tumour and leaving the adjacent parts and structures, by the natural way from the nose or mouth, and so destroying or removing it; (*b*) *radical,* by laying open the area affected by means of a preliminary operation either by opening the nose or by division of the palate or resection of the upper jaws.

One would be likely to refrain from the latter method, which, to say the least, causes serious loss of blood if one has to operate on small tumours, which cause no particular trouble, if the patient is near to the completion of the period of bodily growth, and thus near to the period of involution of the tumour, and, finally, if the general state of the patient's health forbids any serious operation.

The *radical method* is indicated if we must lose no time, owing to threatening complications or dangers, or if the histological examination has assured us of the sarcomatous nature of the new-growth.

In certain cases one could try to place a galvano-caustic snare over the polypus through the nose or mouth, and sever it from its base. This, however, is technically a difficult matter, and does not succeed unless two fingers are inserted behind the soft palate, and are used to guide the snare into the right position, and this can only be under the condition that the tumour is free, not too large, and there are no signs of invasion of the neighbouring cavities. It often happens that we remove only a portion of the tumour by the snare, and if this be the case, the rest must be removed later on in the same way or destroyed by the galvano-cautery.

The most safe, as regards haemorrhage, and the least dangerous, but also the most tedious, method, is removal by *electrolysis.* (See p. 43.) This will be undertaken in patients broken down in health, and if there is a disposition to haemorrhage, *M. Schmidt* uses currents of 30 to 40 milliampéres; *Kuttner*, even 70 to 92. Between the sittings, which last from ten to twenty minutes, intervals must be allowed until the tissue is destroyed and sloughs have been cast off and discharged. The tumour may also be made to atrophy by deeply piercing into it the pointed burner of an electro-caustic apparatus. All these methods may be suitably combined, according to circumstances. As to the preliminary operations above mentioned, we refer to text-books on general surgery.

(b) Struma Retropharyngea (Struma Retrovisceralis sive Accessoria; Retropharyngeal Goitre).—*Etiology and Pathology.*—This consists of a tumour behind the lower section of the pharynx, in the so-called retrovisceral space, arising from a lateral lobe of the thyroid gland, which, in some cases, is not necessarily in connection with the tumour any longer. In this latter case the tumour takes its origin from the outset as an aberrant part of the thyroid or in an accessory thyroid gland.

Symptoms.—The slow growing tumour does not cause trouble until it has arrived at a certain size, and so bulges forwards the posterior pharyngeal wall. Then dysphagia and dyspnoea are the most prominent symptoms. Inflammatory processes may set in, causing severe dyspnoea and hoarseness, which urgently require operation. The tumour, being very vascular, gives rise to haemorrhages, which may be mistaken for haemoptysis.

On pharyngoscopy or hypopharyngoscopy one may observe the posterior wall of the pharynx at the level of the larynx to be bulging forwards on one side only; there a globular tumour can be detected with a smooth and vascular surface, elastic, and *quite insensitive to touch,* and which moves upwards on swallowing. The larynx is displaced, the rima glottidis appears narrowed, the arytenoid cartilages are concealed, as partially also are the vocal cords.* If pressure is exercised on the tumour, one finds *that it can be moved on one side only in the direction where it is in connection with the thyroid gland, and produces, outside the neck, a projection at the level of the thyroid cartilage.*

Diagnosis.—If one only thinks of the possibility of such a tumour, diagnosis should not be too difficult, considering the whole complex of symptoms. Retropharyngeal abscess and other tumours *are movable to both sides.* In the case of a real aberrant goitre, where the palpating finger is not guided by the connecting bridge and the tumour can be moved in all directions, diagnosis might be impossible.

Prognosis.—Owing to serious complications, prognosis is usually doubtful.

Treatment.—If the troubles caused by the tumor are considerable, the tumor must be excised from the outside. If suffocation is threatening, tracheotomy is required. In slighter cases a trial should be made with iodine in the form of thyroid tablets or iodothyrin tablets. Iodo-vasogen (6 per cent.) is also useful in some cases, and I order it for external and internal use: externally, as ointment; internally, 5 drops three times daily. The dose is increased until 15 drops are taken thrice daily after meals in water or milk. Injections into the tumour are harmful and objectionable.

2. MALIGNANT TUMOURS.

The pharynx is not as seldom the seat of a malignant growth as was at one time supposed. Sarcoma, carcinoma, and lymphosarcoma, or, as it is still called, malignant lymphoma, are observed. The latter, on the whole rare, will be separately discussed on account of its peculiarities.

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* The author, throughout this work, uses the term "*vocal lips*" in place of the commonly used term "*vocal cord.*" It is apparently more accurately descriptive of the structures, but for the convenience of the readers of translation, the word "cord" is used.

(a) **Sarcoma and Carcinoma.**—Of the various parts of the pharynx, the tonsils are most commonly the seat of malignant new-growth; more rarely, the deeper parts, viz., the pyriform sinus and posterior pharyngeal wall. Occasionally malignant tumours have been seen arising from the uvula, soft palate, and other parts, but considering the great ramification of the growth, it would be difficult to ascertain the exact origin in any given tumour. Neoplasms of the neighbouring organs, *e. g.*, of the tongue and its root, of the larynx, and of the lymphatic glands, may also grow into the pharynx. Again, we desire to point out that some of the intramural (palatal) growths may show themselves to be sarcomata. Malignant tumours observed in the nasopharyngeal space are often primarily seated in the nose or mesopharynx, and have grown down from there into the throat.

Symptoms and Course.—At the commencement the symptoms are not very pronounced, and only the swollen glands—less in sarcoma than in carcinoma—would point to this ominous disease. But sooner or later definite symptoms do not fail to present themselves, such as disturbances of speech and swallowing, which increase and become aggravated until they cause violent pain. The latter often radiates towards the ear of the affected side. If ulceration has taken place, an offensive smell from the mouth occurs, and it is sometimes this circumstance which compels the patient to consult the physician. Feeding becomes difficult, the patient constantly swallows the foul and putrefying masses, loses weight, and cachexia sets in, and then, with pain and long-drawn-out suffering, the patient dies from exhaustion. In other cases a pneumonia caused by the aspiration of decomposed particles, or a haemorrhage or an attack of dyspnoea, puts an end to the suffering. Death is rarely caused by metastases in other organs.

Diagnosis.—In making the diagnosis one should remember that *sarcoma tends to form a tumour mass*, while carcinoma shows more *tendency to ulcerative destruction.* Carcinoma spreads more into the adjacent tissues, and the glands are early affected (swollen) and form large masses, in comparison with which the primary tumour might be insignificant. Sarcoma has a more smooth surface, is softer and less tuberous or lumpy, than carcinoma. (See Fig. 89.)

Ulcerating new-growth, especially carcinomatous ulcers, can easily be confounded with gummatous or tubercular ulceration. The differential diagnosis between chancre of the tonsil and carcinoma also would often be difficult. Here the same points are available as in analogous diseases of the mouth. In doubtful cases trial with potassium iodide and microscopical and eventually bacteriological examination, would be of great service. Unfortunately, round-celled sarcoma is very like a gumma under the microscope. A sarcoma of the tonsil, softened in the centre, sometimes conspicuously resembles a peritonsillar abscess.

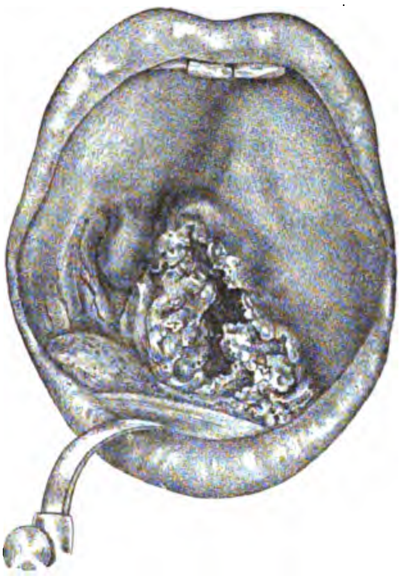

Fig. 89.—Ulcerating carcinoma of the left tonsil (*Türck*).

Prognosis.—If it is possible to remove the tumour and all the affected glands radically, prognosis might be fair, but we must not be too rash, for recurrences are very frequent in spite of radical and successful operation (*Kroenlein*).

Treatment.—If the disease has been recognised early, a cure may be possible, as, for instance, by amputation of the uvula or tonsil. The operation will be very difficult if the tumour is large and the regional glands are affected. As to the required operation, see text-books on general surgery. Inoperable cases may be treated by electrolysis or the galvano-cautery; and in some cases arsenic in increasing doses, and eventually subcutaneously, has been of some benefit; at least, various authors have reported good results. Taken all in all, these measures are only palliative and may render the patient's condition more bearable, at least for a time; but often, not even this. Will radium, perhaps, properly applied, or the *x*-rays, prove a success in the future? In the advanced stages of the disease the physician has a very difficult and sad task to fulfil. The pain may be relieved by painting with cocaine, and the horrible foetor diminished by deodourizing gargles. Later the oesophageal tube and nourishing enemata will have to be used, and gastrostomy performed in order to prevent the unfortunate sufferer from dying of starvation and from suffocation, which will entail a tracheotomy, or both operations may have to be performed. Finally, life may be rendered supportable, sit venia verbo, by injections of morphine.

(b) Lymphosarcoma (Malignant Lymphoma).—*Etiology and Pathology.*—Lymphosarcoma originates in the lymphatic tissue of the pharynx (tonsils and lymph-follicles); the tumour usually arises from the pharyngeal tonsil, according to *Störk*, and also from the uvula, the palatine arches, and the epiglottis. Metastases are likewise met with only in lymphatic organs (glands, spleen, and intestines). Histologically, lymphosarcoma shows masses of small round-cells (lymphocytes), which are contained within the meshes of a very densely reticulated connective tissue. It can be distinguished from the normal adenoid tissue by its homogeneous structure and the absence of lymph-follicles. Thus it shows the same structure as round-celled sarcoma. In what relation lymphosarcoma stands to leukaemia and pseudo-leukaemia may be questioned. *Mikulicz* suspects the lymphosarcoma to be a special pharyngeal form of pseudo-leukaemia.

Symptoms.—The symptoms are those of a softened and ulcerated tumour. The lymphatic glands of the neck are swollen at an early stage of the disease. Curiously enough, some of these swellings are liable to shrink afterwards spontaneously, but usually, and soon after, recurrences follow or fresh tumours occur in the neighbourhood or in a more distant region. The patient succumbs exhausted or dies from suffocation or intracranial lesions.

Diagnosis.—The diagnosis is not always easy at the beginning. Mistakes with adenoid vegetations, or if ulceration has already taken place, with gummatous or carcinomatous ulcers, are often made. The syphilitic nature of a growth might perhaps be ascertained by the effect of large doses of iodide of potassium. Carcinoma and sarcoma can be histologically distinguished from lymphosarcoma, but not so the round-celled sarcoma.

Prognosis.—These cases of lymphosarcoma, which, fortunately, are of rare occurrence, are very grave, for relapse and metastasis cannot be prevented in spite of all therapeutic or spontaneous retrogressions.

Treatment.—Extensive operations, owing to the unfavourable prognosis, have no sense or justification, but it might be well, for the sake of the temporary relief, to excise one or other part of the tumours. In any case arsenic should be given internally or subcutaneously, as very good results and improvement have been observed to follow; also *Röntgen* rays and radium might be applied where the circumstances permit.

X. NERVOUS DISORDERS.

1. DISORDERS OF SENSIBILITY.

(a) **Anaesthesia.**—(1) *Central causes* of pharyngeal anaesthesia are: Hysteria (mostly unilateral), basal meningitis, gumma of the basis cranii, disseminated sclerosis, bulbar paralysis, haemorrhage, locomotor ataxia, tumours. (2) *Peripheral causes* are: Diphtheria; artificial anaesthesia locally produced, *e. g.*, by cocaine, alypine, menthol, or by the internal (subcutaneous) use of bromides, chloral, and morphine. Anaesthesia does not usually cause any trouble, save for a certain peculiar sensation of roughness in the throat. If, on the other hand, the pharyngeal muscles or larynx are also paralysed, then the subjective symptoms are really very considerable. In this case the danger of misswallowing and from the pneumonia caused by it is very urgent.

(*b*) *Hyperaesthesia* is often produced by chronic catarrh, and may be frequently observed in smokers or drinkers, but also in persons who neither smoke nor drink, and whose hypersensitiveness of the throat, though unaccompanied by catarrh, is the sign of a general nervousness or neurasthenia. Besides the hyperaesthesia, there also exist other reflex disturbances which may be brought out by touching certain sensitive spots.

(*c*) *Paraesthesia.*—Various forms of paraesthesia are found in chronic catarrh, especially in association with tonsillar concretions or calculi, or in the dry form of pharyngitis (pharyngitis sicca), and cause the most perverse sensation (see p. 278). Paraesthesia is often symptomatic of hypertrophy of the lingual tonsil (see p. 218), and after injuries by foreign bodies (see p. 318). In some cases it is an early sign of pulmonary tuberculosis; it is also frequent in hysteria (globus hystericus), anaemia, and chlorosis. The patients themselves report the most curious things as to the site and character of their most unpleasant sensations, and many become real hypochondriacs. The anatomical changes, moreover, are frequently insignificant or are altogether missed. In the latter case the probe will help us a good deal in finding out the spot or region of the abnormal sensation—a very tedious and difficult business.

Treatment must in all cases be directed against the causes of the disease. Local treatment (treatment of the existing catarrh, removal of tonsillar concretions or hypertrophied tonsils, etc.) must be aided by all general measures (diet, change of air, hydropathic cures, tonic and antisyphilitic treatment).

In anaesthesia and paraesthesia the electric (galvanic or faradaic) current is useful. In hyperaesthesia the local application of cocaine is sometimes required, but we should not be too liberal with the use of cocaine; and menthol lozenges, for the most part, are quite satisfactory. In purely nervous hyperaesthesia the galvano-cautery or cauterisation with chromic or trichloracetic acids proves very useful. Whether these remedies destroy small foci, or whether they act as powerful suggestive agents, is open to question. In some other hyperaesthetic cases also, which perhaps is nothing else than a variation of paraesthesia, cauterisation is very successful.

2. DISORDERS OF MOBILITY.

(a) **Paralysis.**—We are here chiefly concerned with the paralysis of the soft palate, which is either complete or partial, bilateral or unilateral.

Etiology.—Paralysis of the velum palati is either central or peripheral.

(1) *Central paralysis* occurs mostly in grown-up persons, and is caused by inflammatory or degenerative processes in the brain, medulla oblongata, or spinal cord, *e. g.*, progressive muscular atrophy, amyotrophic lateral sclerosis, tabes, bulbar paralysis (paralysis glosso-labio-pharyngea), basal meningitis (gumma), and syphilitic disease of the brain (gumma and endarteritis obliterans, etc.).

(2) *Peripheral paralysis* is frequently due to *diphtheria*, and is chiefly met with in children or juvenile patients, but it has occurred also after *influenza*. In facial paralysis also the velum palati may be paralysed if the seat of the lesion is at or above the geniculate ganglion (the motor nerves of the velum palati run with the great petrosal nerve (nervus petrosus superficialis major), which is a branch of the geniculate ganglion and helps to form the Vidian nerve (n. vidianus), the latter being a branch of the facial (seventh cranial), to *Meckel's* ganglion (spheno-palatine ganglion). Sometimes paralysis of the vagus (inferior laryngeal nerve) is associated with paralysis of the soft palate, which goes to show that the vagus, or at least some of its filaments in the pharyngeal plexus, has a share in the supply of the palatine muscles.

From pure mechanical reasons inflammatory processes are liable to more or less disturb the mobility of the soft palate; for instance, tonsillar and peritonsillar abscesses, collateral oedema, and also adenoid vegetations, tumours, or enlarged tonsils.

Symptoms.—In marked paralysis the nasal cavity is not shut off during speech, which, therefore, becomes strongly nasal (rhinolalia aperta); fluids regurgitate through the nose, and all functions or actions, which can only be performed if the soft palate is undisturbed in its movements, are more or less inhibited, as, for instance, gargling, blowing out the cheeks, sucking, etc. Insufficient action of the palatine muscles, which, as we know, also act as dilators of the Eustachian tube, lead to contraction or occlusion of the tube and to serous transudation into the middle ear, consequent on the diminution of the air-pressure in the cavum tympani, as in hydrops ex vacuo. (See p. 35, et seq.)

I recently had a young woman under treatment who got a paralysis of the soft palate after influenza, and consequently a bilateral exudative catarrh of the middle ear, which subsided along with the paralysis. Examination showed a great impairment of hearing, and behind the drum one could distinctly see bubbles in the contained fluid.

The pharyngoscopic examination is very characteristic. In unilateral paralysis the velum palati is asymmetrical. The affected side drops, and is lower and more forward than the other, and the arcus is less curved. On intonation the paralysed side, together with the uvula, is drawn towards the healthy side; in bilateral paralysis the soft palate hangs flaccidly down and is immobile. In paresis all these symptoms are less prominent.

Paralysis of the velum palati is usually combined with that of the pharyngeal constrictors, which can be recognised by the mucous membrane remaining smooth, even during swallowing or retching. If the lower part of the constrictor is paralysed, food cannot be swallowed and sticks at the root of the tongue in front of the epiglottis, sometimes causing not inconsiderable dyspnoea, and has to be coughed out or raked up with the finger. If, in addition, the other pharyngeal muscles are paralysed, as is so often the case in progressive muscular atrophy or bulbar paralysis, the epiglottis cannot be shut down during swallowing, and food is liable to pass into the larynx. Another complication in bulbar paralysis is the paralysis of the tongue, which aggravates still more or even inhibits articulation and the taking of food.

Diagnosis.—The diagnosis can be easily made if one takes into consideration the whole clinical picture. An incomplete paralysis may be easily overlooked if the nasal speech is the only symptom. The cause of the paralysis is in many cases rather difficult to ascertain. If the cause is an acute infectious disease, especially if the patient has suffered from diphtheria or influenza, we can then assume, without hesitation, a peripheral paralysis due to neuritis. In *peripheral paralysis,* moreover, the nerves and muscles, after one or two weeks, show the typical reaction of degeneration, *i. e.,* they do not react at all to faradic and very little, or not at all, to galvanic stimulation, and undergo atrophy. *In central paralysis,* on the other hand, the electric irritability is undisturbed and no atrophy supervenes. That a paresis might be simulated by a mechanical impediment, as the result of local inflammatory and other processes, has already been mentioned.

Prognosis.—The prognosis is favourable in peripheral paralysis and unfavourable in a central lesion, save from syphilitic or hysterical diseases.

Treatment.—If we have reason to suspect syphilis, antisyphilitic treatment must be applied, *i. e.,* a combined cure with mercurial ointment and potassium iodide (unguentum cinereum, 4 grammes per diem); and potassium iodide, 15 to 200 (one tablespoonful three times daily). Peripheral paralyses, especially postdiphtheric paralyses, recover, after a time, without treatment, but, better still, and quicker under roborant diet, which may be greatly assisted by brine baths and electric treatment. The electric current is generally, and in all cases of paralysis, very useful. The anode is put upon the neck or chest; the button-shaped cathode, which must be so constructed that the current may be opened or closed according to the will of the operator, on the paralysed muscle, after previous cocainisation, if this is required. In very nervous and resistant patients both electrodes, fitted with a broad pad, are put outside on the neck. In hysteria electric treatment works wonders, but improvement is due more to suggestion than to the current, and in some cases suggestive or hypnotic treatment may be resorted to. In complete paralysis we must feed the patient by an oesophageal tube.

(b) Cramps (Spasms).—Irritative motor lesions cause tonic or clonic spasms or rhythmic twitching (contractions); in many cases all the pharyngeal muscles and those of the neighbouring organs, viz., tongue, larynx, oesophagus, and ear, are attacked by cramps, and very rarely only one single group of muscles.

Etiology.—Occasionally transient tonic spasms of the constrictors and muscles of the Eustachian tube are excited if we insert a catheter into the tube; also, a morsel of food swallowed too hastily is liable to cause spasms in the pharynx or gullet. In very nervous and excitable (hysterical) patients inflammatory processes, *e. g.*, pharyngitis lateralis or hypertrophy of the lingual tonsil, are apt to excite spasms. Central causes are principally *hysteria and rabies.* In rabies (hydrophobia) tonic spasms are reflexly set up at each endeavour to swallow, and even at the mere sight of water. Clonic spasms are not infrequently caused by irritation of the trigeminal nerve. Where and how the nerve is irritated often escapes our knowledge. Among other causes of clonic spasms may be mentioned: hysteria, paralysis agitans, disseminated sclerosis, poisoning by lead, mercury, and alcohol, disease of the occipital lobes of the brain, and occasionally clonic spasms are associated with cramps of the facial muscles (tic convulsif).

Symptoms.—In tonic spasm of the pharyngeal constrictors *dysphagia* is very marked. The morsel of food sticks fast in the throat or gullet, and causes a painful sensation behind the sternum, and regurgitation frequently occurs. "*Globus hystericus*" is probably nothing else than a tonic contraction of the oesophagus and pharynx, moving up and down, and is excited by psychic or local irritation (hypertrophy of lingual tonsil, pharyngitis, etc., see p. 218).

In tonic spasm of the velum palati the soft palate is firmly pressed against the posterior pharyngeal wall, and the Eustachian tube, in consequence of the tonic contraction of the levator palati, remains open, and the patient hears his own voice much louder than usual (autophony).

In clonic spasms the velum palati makes jerky movements, and the patient and his listeners hear a peculiar clicking or crackling noise, caused by the quick alternations of opening and closing of the tube. In such a case one may see the tympanic drum moving alternately, backwards and forwards, that is, alternately retracting and bulging outwards.

Tonic spasms do not usually last long, but the clonic spasms, on the other hand, are very persistent, although they are not usually the cause of much complaint by the patient.

Diagnosis.—The diagnosis of clonic spasms is mostly easy. The tonic spasms are liable to be mistaken for fibrous stricture. But while in the case of tonic spasm of the constrictors transient narrowing is caused, in stricture, however, the narrowing of the lumen is permanent, so that in spasm a bougie may be introduced, without hindrance, into the stomach during the free interval, but in the case of stricture the insertion of the oesophageal probe will always be difficult. Hysterical spasms may be recognised by the presence of other signs of hysteria.

Prognosis depends on the true cause of the spasms.

Treatment.—In treatment also the cause will be our guide. The reflex excitability may be diminished by bromides and other sedatives. The diet must be regulated, and it should be fluid, sloppy, and not too warm. In long-persisting spasms feeding through an oesophageal tube may be required.

PART IV.

Diseases of the Larynx and Trachea.

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Diseases of the Larynx and Trachea.

GENERAL SECTION.

I. ANATOMY.

The larynx is formed by a skeleton of cartilages suspended from the hyoid bone, and connected with one another by ligaments, and rendered movable by muscles. When at rest, the larynx lies between the upper margins of the third and the lower margin of the sixth cervical vertebrae. The upper aperture, covered by the laryngeal lid (epiglottis), communicates with the laryngeal part of the pharynx (aditus laryngis). The laryngeal part of the pharynx (hypopharynx) is situated behind the larynx, so that its anterior wall forms the posterior wall of the larynx. (See p. 237.) The lower aperture leads into the trachea, with which the larynx is continuous. On both sides of the larynx are situated the lower hyoid muscles (mm. sterno-, thyro-, omo-, hyoidei), all of which are ensheathed by the deep fascia of the neck (fascia colli); the large vessels and nerves of the neck are found still more laterally. The thyroid gland also partially covers its lateral wall, and not infrequently a middle lobe (pyramid), arising from the gland, passes upwards in front of the larynx. The anterior wall of the larynx is covered by the median fibers of the above-named muscles, but in the middle line itself the larynx is only covered by two laminae of the fascia colli and the skin.

The Cartilaginous Skeleton.—(*a*) *Cricoid Cartilage.*—The base is formed by the *signet-ring-shaped cartilage* (cartilago cricoidea, cricoid cartilage), the broader posterior plate of which has two articular facets on its upper margin for articulation with the two pitcher-shaped cartilages, and near the lower margin on either side one facet for articulation with the inferior cornua of the thyroid cartilage. The thinner arc of the cricoid cartilage is directed forwards.

(*b*) *The Thyroid Cartilage.—The shield-shaped* or thyroid cartilage (cartilago thyroidea), which by its size and configuration outlines the shape of the larynx, consists of two plates (alae or wings), united in front at an acute angle. Its upper border is irregularly curved and is notched in the middle line by the easily palpable incisura thyroidea. This part projects, especially in thin men, more than in women, and is known as the prominentia laryngea or Adam's apple (pomum Adami). The posterior vertical border of the thyroid plates ends above in the superior cornua, and below, in the inferior cornua.

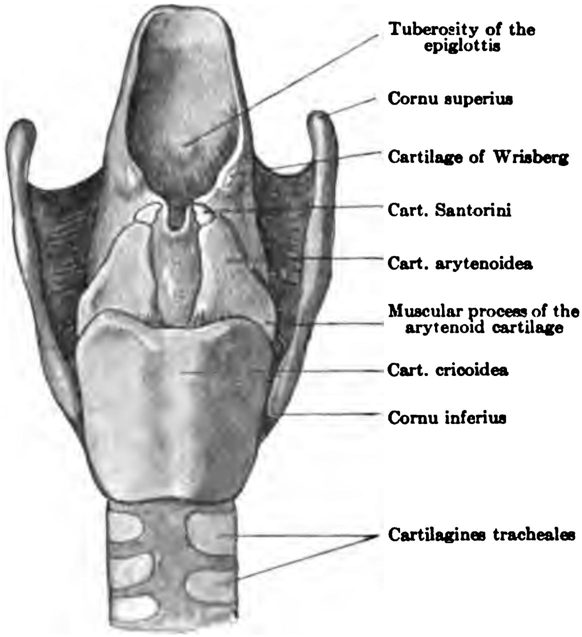


Fig. 90.—The larynx, viewed from behind, the muscles being removed.

(*c*) *The Epiglottis.*—The lid (epiglottis) is attached by its petiolus or apex to the margin of the incisura thyroidea, and forms there a pad-like projection towards the lumen of the larynx, which is spoken of as the tuberculum epiglottidis or epiglottic cushion or pad. The broad and free upper margin of the epiglottis is directed backwards, and varies much in shape, a circumstance of great importance during laryngeal examination. (See Fig. 90.) Besides these three single, there are also three paired, cartilages.

(*d*) *The Arytenoids.*—The pitcher cartilages (arytenoid cartilages, cartilagines arytenoideae) are the most important as regards the functions of the larynx, because they dilate or narrow the rima glottidis, according to their position. They have the shape of small, three-sided pyramids, with their apices directed upwards and a little backwards, and their bases articulating with the posterior plate of the cricoid cartilage (see above). Of the three surfaces, one looks inwards, another looks outwards, and the third looks backwards. The base of the pyramid ends in front as the *vocal process;* and outwardly and posteriorly it becomes the *muscular process*, giving attachment to the mm. crico-arytenoideus lateralis and posticus. The *vocal process* (anterior angle), however, which is covered with mucous membrane and is of a yellowish, glistening colour, has attached to it the true vocal cord (and the m. thyro-arytenoideus).

(*e*) *Cartilagines Santorini et Wrisbergii.*—On the apices of the arytenoid cartilages, and attached to them, are the small *cartilagines Santorini* (cornicula laryngis or cartilagines corniculatae), and situated somewhat more laterally, the *cartilagines Wrisbergii* (cartilages of *Wrisberg* or cuneiform cartilages). Both pairs are contained in the aryteno-epiglottidean folds of mucous membrane (plica aryepiglottica) which bound laterally the aditus laryngis, leaving a small notch free posteriorly, called the posterior commissure (incisura seu pars interarytenoidea). The posterior commissure, on phonation, appears as a small chink, and on quiet respiration, when the arytenoid cartilages separate themselves as a flat groove which corresponds to the posterior laryngeal wall (see p. 348).

(*f*) *Sesamoid Cartilages.*—Still more insignificant than the cartilages of *Santorini* and *Wrisberg* are the *sesamoid cartilages*, which sometimes—not always—are found on the lateral margin of the arytenoid cartilages and on the anterior end of the vocal cords, and are here of a yellowish luster. (See Fig. 91.)

Ligaments of the Larynx.—The membranous and ligamentous apparatus of the larynx is, owing to the complicated function of this organ, equally complicated. (See Fig. 92.) Between the hyoid bone and thyroid cartilage are the thyrohyoid ligaments (lig. thyro-hyoideum), with a broad middle portion and two narrower lateral portions, the latter being attached to the superior cornua of the thyroid cartilage. The space between the lateral and middle thyroid ligaments is filled in by the thyrohyoid membrane (membrana thyrohyoidea), which is perforated by the superior laryngeal nerve and artery. The lateral ligaments each contain a nodule of cartilage—the cartilagines triticeae.

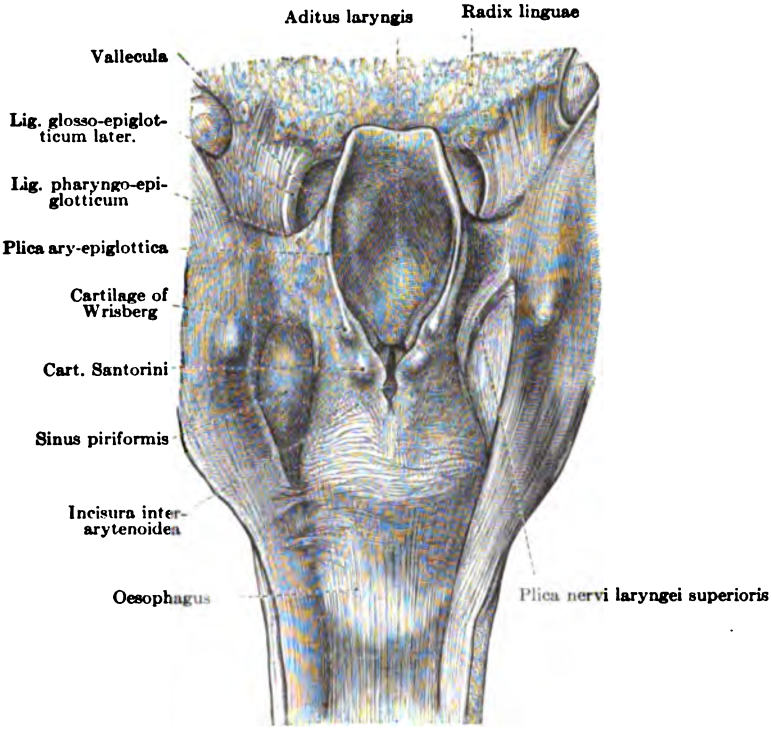


Fig. 91.—The aditus laryngis, viewed from behind. The posterior wall of the pharynx is cut lengthwise and turned outwards (after *Toldt*).

Between cricoid and thyroid cartilage passes the strong elastic lig. crico-thyroideum medium s. conicum. The arteria cricothyroidea runs in front of and across the lig. conicum, which fact is of surgical importance. Frequently a small lymphatic gland and a median lobe of the thyroid gland or the remains of such a lobe may be found lying on the ligament. Between the cricoid cartilage and trachea extends the lig. crico-tracheale. The lig. aryteno-epiglottica are not true ligaments, but only reduplications of the mucous membrane; so also are the lig. pharyngo-epiglottica (see p. 238) and lig. glosso-epiglottica (p. 169), which, respectively, connect the epiglottis with the lateral pharyngeal wall and root of the tongue. All these last are, therefore, called *plicae*, or folds.

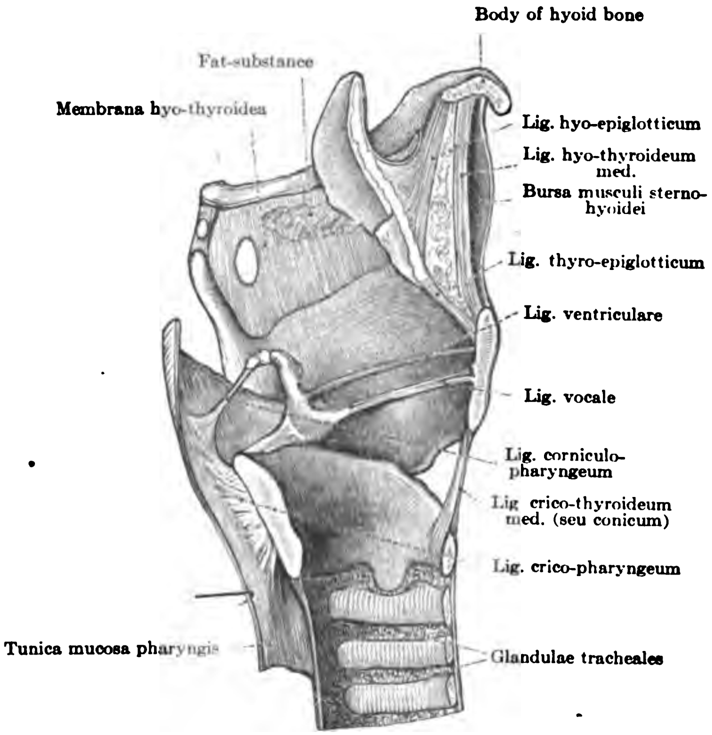


Fig. 92.—Ligaments of the larynx. The left half of the larynx, viewed from within (*Toldt*).

The capsules of the various joints are strengthened by numerous small ligaments, and only the joint between the cricoid and arytenoid cartilage, by which the arytenoid cartilage rotates around a vertical axis for the dilatation and narrowing of the rima glottidis, remains quite loose and flaccid.

The *vocal cords* (lig. vocalia s. thyro-arytenoidea inferiora) and the *ventricular ligaments* (lig. ventricularia s. thyro-arytenoidea superiora) are of special importance. The *vocal ligaments* (vocal cords) are dense elastic ligaments running sagittally on each side from the vocal process of the arytenoid to the inner surface of the thyroid cartilage. Immediately above them are the *ventricular ligaments*, which are also called "false vocal cords." Both pairs of ligaments are covered with mucous membrane, which forms a small pouch between them, called ventriculus or sinus *Morgagni*, continued upwards into the appendix ventriculi. (See Figs. 93 and 94.)

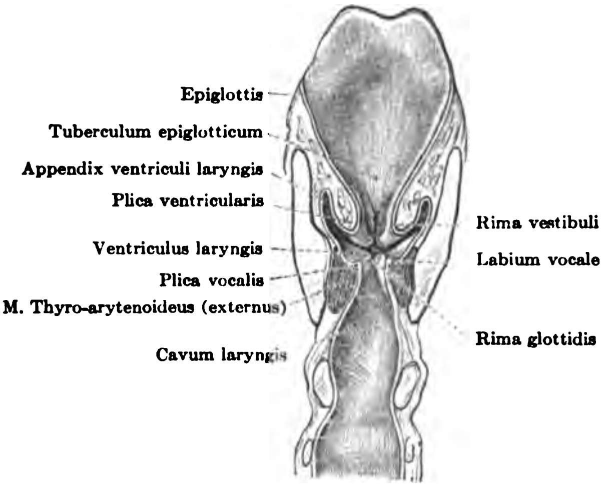


Fig. 93.—Frontal section of the larynx, viewed from behind (*Toldt*).

The ligamentum vocale (lig. thyro-arytenoideum inferior), together with the *vocal muscle* and the covering fold of mucous membrane (plica vocalis), forms a prismatic structure which projects sharply into the lumen of the larynx, and is now generally called the labium vocale (vocal cord). The space between both vocal cords is called the rima glottidis, or, shortly, "glottis." The angle formed by the anterior attachment of both vocal cords to the thyroid cartilage immediately below the epiglottic tubercle (epiglottic pad) is known as the anterior commissure. The anterior portion of the "glottis," situated between the *vocal cords,* is called pars membranacea, and the posterior part, situated between the arytenoid cartilages, is called the pars cartilaginea. On phonation, both vocal cords approach each other, and the rima glottidis forms a straight chink. During respiration, however, the vocal cords separate from each other, and the rima glottidis now forms a triangle, the apex of which is directed towards the anterior commissure, with the base towards the posterior laryngeal wall. The sides of this triangle are not straight lines, however, but show a more or less marked curvature, according to the shape of the vocal processes. The *ventricular folds* (plicae ventriculares), which are situated immediately above the *vocal cords,* are thick pads,

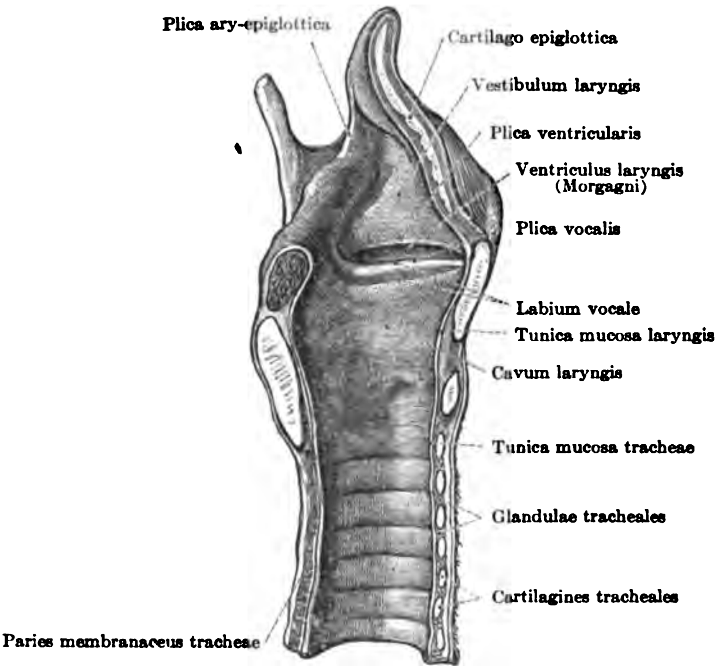


Fig. 94.—Sagittal section of larynx left side (*Toldt*).

likewise containing ligamentous and muscular fibers though much less than the vocal cords, form the rima vestibuli. This latter is the border line between the upper and middle parts of the larynx. All that portion of the larynx which lies above the rima vestibuli is called the vestibulum, and all that which lies below the vocal cords is called the regio subglottica (the whole vocal apparatus is sometimes also called the "glottis").

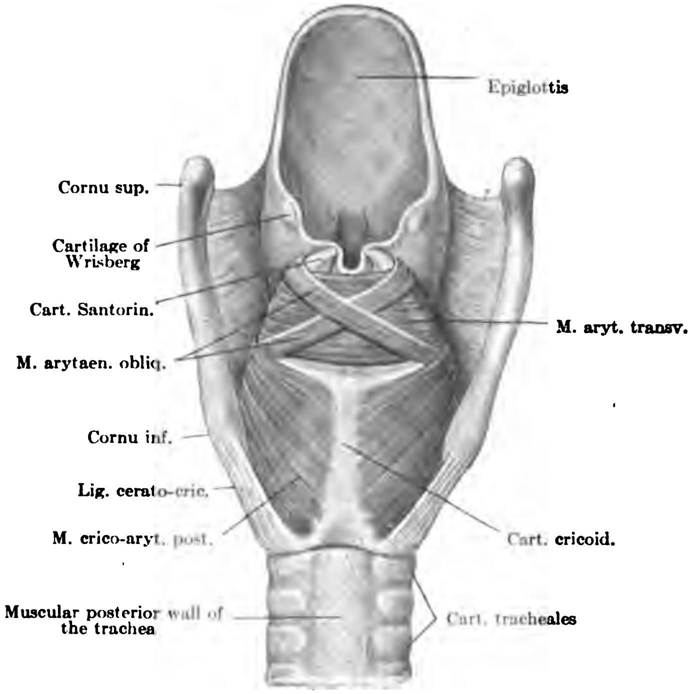


Fig. 95.—Muscles of larynx from behind (after *Störk*).

The muscles of the larynx (see Figs. 95 and 96) are divided, according to their function, into two groups:

(*a*) *Muscles which move* or *fix* the larynx as a whole. To this group belong the muscles below the hyoid bone (sternohyoid, omohyoid, sternothyroid, thyrohyoid), and the inferior constrictor pharyngis (also m. laryngo-pharyngeus), whose fibers embrace the thyroid and cricoid cartilages posteriorly. These muscles act either as depressors or elevators of the larynx.

(*b*) Muscles which, by their action, *regulate the width of the glottis* and *tension of the vocal cords* and also exercise influence upon the position of the epiglottis, *configurating the aditus laryngis*. To these belong:

1. *Dilators of the* glottis (abductors).

2. *Narrowers* of the glottis and simultaneously configurators of the aditus laryngis (adductors).

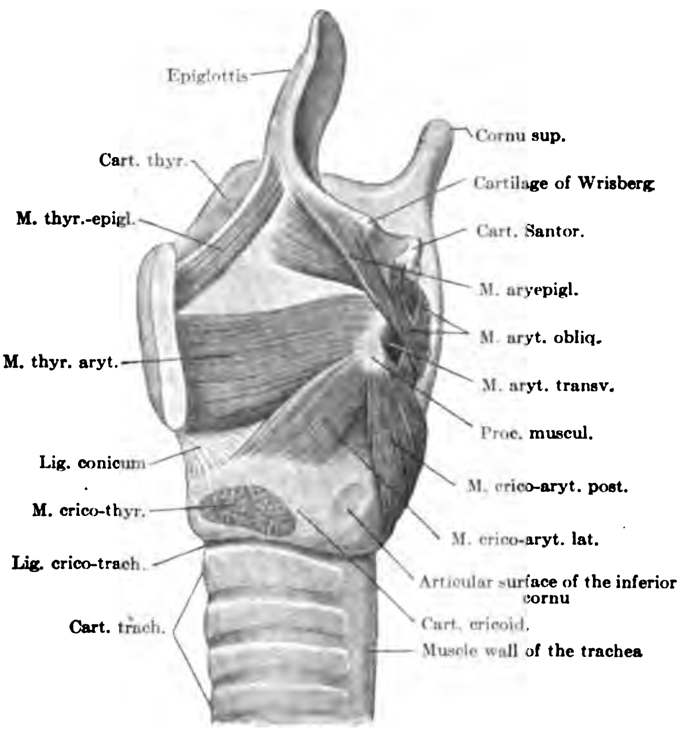


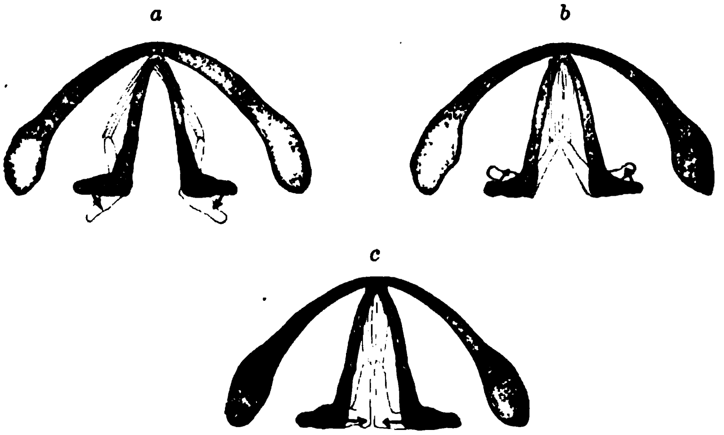
Fig. 96.—Muscles of larynx, lateral view. The left part of the thyroid cartilage is removed (*Störk*).

3. *Tensors* of the vocal cords.

1. *Dilator* (*opener*) *of the glottis* (abductor). There is only one muscle which does this—the m. crico-arytenoideus posticus, called shortly "posticus." It arises from the posterior surface of the cricoid plate, and passes outwards and upwards to be inserted into the muscular process (postero-external

angle) of the arytenoid cartilage. Its action is to rotate the arytenoid cartilage round a vertical axis in such a manner that the muscular processes are drawn backwards, thereby moving the vocal processes (anterior angles) outwards; the glottis is by this action widened into a triangular space.

2. *Narrowers of the glottis* (*adductors*) are chiefly the mm. crico-arytenoideus lateralis, shortly called "lateralis," and the m. arytenoideus. *The m. crico-arytenoideus lateralis* arises from the upper border and lateral surface of the arch of the cricoid cartilage, and passes upwards and backwards to be inserted into the muscular process of the arytenoid cartilage. Its action is to draw the muscular processes forwards, thereby rotating the vocal process inwards and narrowing or closing the glottis. It is thus the antagonist muscle of the "posticus."



a, Action of posticus; *b*, action of lateralis; *c*, action of transversus.

Fig. 97.—The thyroid and arytenoid cartilages are cut horizontally. The action of the muscles is marked by the black arrows, the effect upon the glottis by red lines.

The m. arytenoideus consists of two portions, one single, the fibers of which run transversely from the posterior surface of one arytenoid cartilage to the posterior surface of the opposite cartilage, and so filling the gap between the two cartilages; it is, therefore, also called m. interarytenoideus or "transversus." Behind this muscle the fibers of the m. arytenoideus obliquus, shortly called "obliquus," cross obliquely, and which arise on both sides from the muscular processes of the arytenoid cartilage, to run obliquely across to the other side, and to be inserted into the aryepiglottic fold and the epiglottis (m. aryepiglotticus) (see Fig. 97).

Action of the transversus: approximates the arytenoid cartilages, thus closing the back part of the glottis. (To close the entire glottis the lateralis and transversus must act together.)

Action of the obliquus: aids the transversus in closing the glottis, constricts also the entrance to the larynx, and depresses the epiglottis, and is, therefore, also called the constrictor vestibuli laryngis. Of special importance is the m. thyro-arytenoideus, which is fixed between the thyroid and arytenoid cartilages, and is parallel to the vocal cords. It consists of two portions—(1) Of the three-edged, prismatic, thyro-arytenoideus internus, shortly called "internus," situated within the "vocal cord," the *m. vocalis*, properly so-called, and (2) m. thyro-arytenoideus externus, whose fibers unite with the inner portion and the "lateralis." The "*internal*" *portion* is inserted into the *vocal process*, the "*external*" portion into the *muscular process*. The action of the m. thyro-arytenoideus is not yet satisfactorily explained. The external portion, which shows some variation and joins the "lateralis," draws the muscular processes of the arytenoid forwards and thus helps in closing the glottis. The internus also takes part in this action by shortening (*i. e.*, broadening) the vocal cords, which are thus brought closer to each other. At the same time, by its graduated contractions, it brings about the exact intonation of the voice and various registers (see next chapter). This action is probably aided by fibers which belong to the superior thyro-arytenoid ligaments (m. thyro-arytenoideus superior or ventricularis).

3. *The tensor of the vocal cord* is the m. crico-thyroideus, shortly called "anterior." It arises from front and side of the cricoid arch, and is inserted in a fan-like manner into the lower and inner border of the thyroid cartilage and inferior cornu.

Its action is to make the vocal cord tense by elongating the distance between its too fixed extremities.

How this elongation is produced is still a matter of controversy. One opinion is that the thyroid cartilage, as the movable part, is rotated around a transverse axis in such a way that its anterior part is moved downwards and towards the cricoid cartilage. Another suggestion is that the thyroid cartilage, being fixed by the muscles of the neck attached to it, the cricoid cartilage is moved around a transverse axis in such a manner that its anterior part (arc) is approximated upwards to the thyroid, while its plate (posterior part), together with the arytenoid cartilages, moves downwards. In both cases the thyroid and arytenoid cartilages are separated from each other and thereby the vocal cords are made tense.

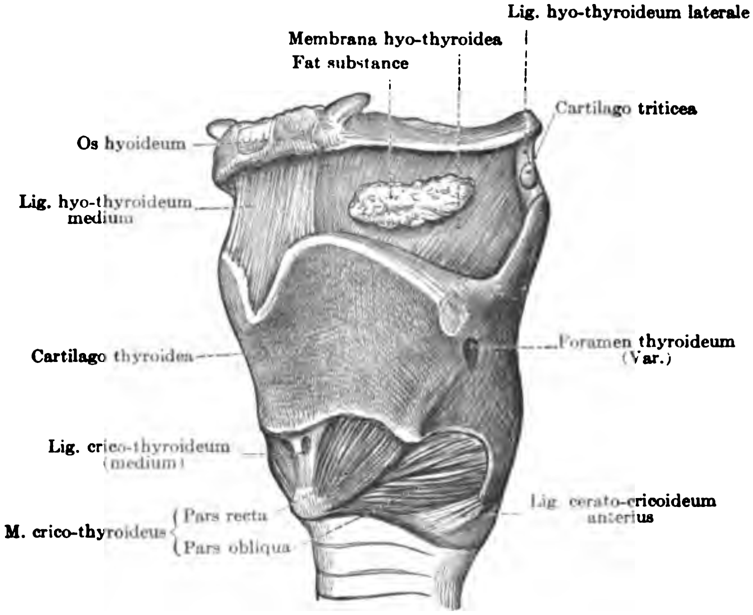

Lig. hyo-thyroideum laterale
Membrana hyo-thyroidea
Fat substance
Cartilago triticea
Os hyoideum
Lig. hyo-thyroideum medium
Cartilago thyroidea
Foramen thyroideum (Var.)
Lig. crico-thyroideum (medium)
Lig. cerato-cricoideum anterius
M. crico-thyroideus
Pars recta
Pars obliqua

Fig. 98.—M. crico-thyroideus, viewed from in front and left side (*Toldt*).

The course of its fibers would suggest that perhaps the m. crico-thyroideus exercises a pressure upon each side of the thyroid cartilage, with the result that the transverse diameter of the larynx becomes shortened, and consequently the sagittal diameter is elongated; this would, of course, also produce a separation of the thyroid and arytenoid cartilages, and thus a tension and approximation of the vocal cords. The tension of the vocal cords is augmented to a certain degree by strong contraction of the mm. crico-arytenoidei postici.

The action of the laryngeal muscles can thus be brought into the following scheme:

Narrowing or closing (adductor):	M. crico-arytenoid: lateralis. M. arytenoideus transversus et obliquus. M. thyro-arytenoid: externus and internus. M. crico-thyroideus.
Dilating (abductor):	M. crico-arytenoideus posticus.
Tensing:	Crico-thyroideus. M. crico-arytenoideus posticus.

It is absolutely necessary, for the knowledge of certain pathological conditions, to know exactly the action of the various laryngeal muscles. Under *physiological conditions* isolated action of a single muscle does not come into consideration, as *several muscles always* act together in association, either in concert or as antagonists, as is the case in movements of the vocal cords).

The Nerves of the Larynx (See Fig. 99).—The sensory as well as motor nerves are derived from the pneumogastric. Various authors, however, maintain that the motor nerves arise from the *spinal accessory*, viz., from that portion of the nerve which joins the vagus after the exit from the jugular foramen (ramus internus accessorii; the external ramus supplies the mm. sterno-cleido-mastoideus and trapezius).

The *vagus* supplies the larynx by two branches—superior and inferior laryngeal nerve.

The superior laryngeal nerve divides into an internal and an external branch; the internal branch enters the larynx together with the superior laryngeal artery by an aperture in the thyrohyoid membrane (see p. 344), and runs underneath the mucous membrane of the pyriform sinus (plica nervi laryngei: see p. 238); it is the sensory nerve of the mucous membrane which is particularly sensitive, especially in the pars interarytenoidea. The external branch supplies the m. crico-thyroideus;

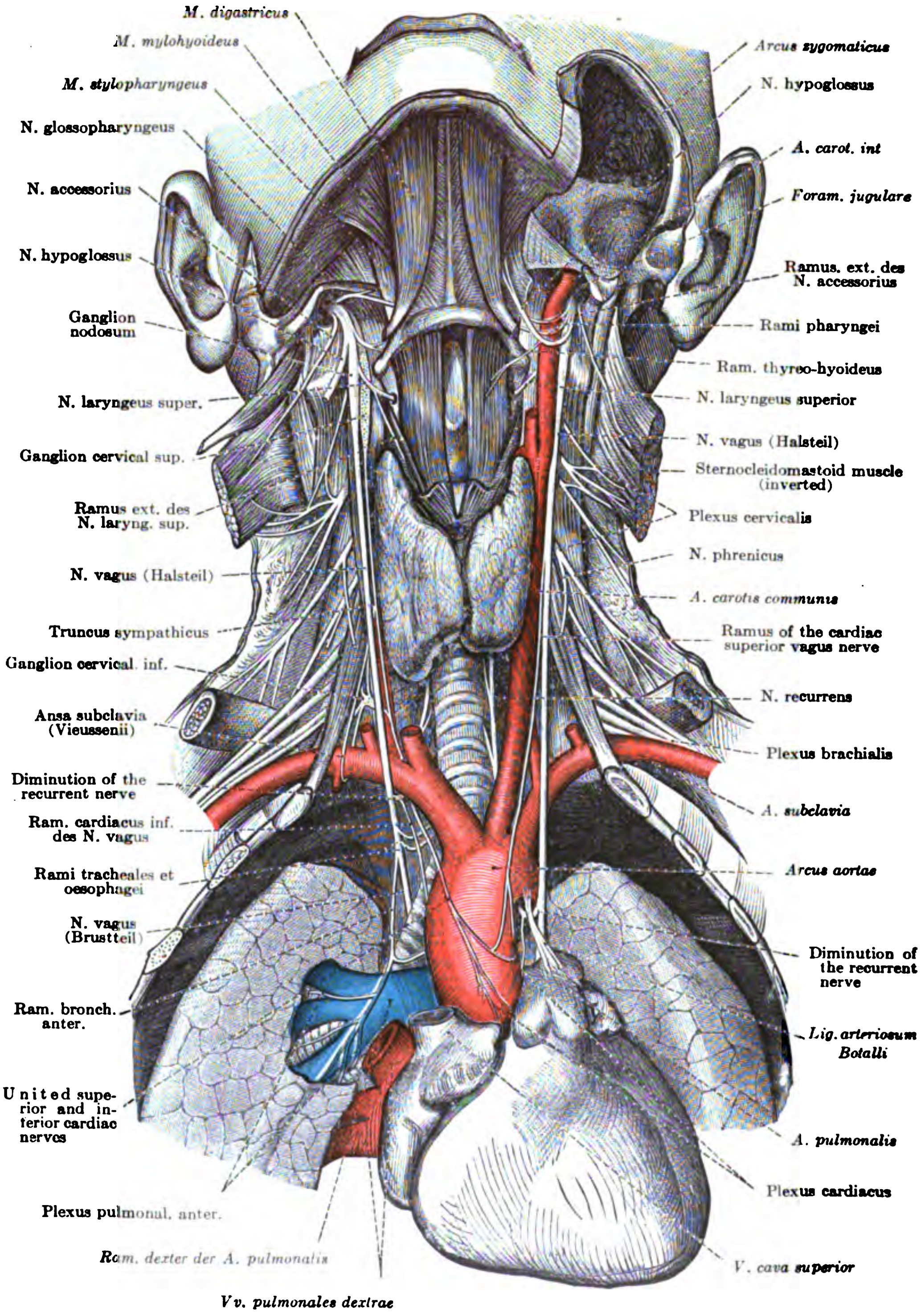


Fig. 99.—Nerves of the larynx (*Toldt*).

but sometimes the latter muscle is also supplied by the recurrent laryngeal nerve (inferior laryngeal).

The internal branch communicates by the *plexus Galeni* with the inferior laryngeal, thus supplying with sensory nerves the deeper parts of the larynx and the upper part of the trachea.

The inferior laryngeal nerve (*recurrent laryngeal, nervus recurrens*), which is very important in the pathology of laryngeal paralyses, arises, on the right side, from the vagus at the level of the apex of the lung, just below the subclavian artery, and winds round that artery from before backwards; on the left side, the recurrent arises further down, below the arch of the aorta, and winds around it from before backwards. Both nerves ascend along the groove between the trachea and the oesophagus, to reach the lower posterior surface of the larynx, and are distributed to all the laryngeal muscles except the crico-thyroideus (see above), giving off, in their course, cardiac, oesophageal, tracheal, and pharyngeal branches, and, finally, communicate with the superior laryngeal nerves (see above).

The different course on the two sides of the body explains why the right recurrent is so often involved in diseases of the apex of the lung and subclavian artery, while the left is often implicated in diseases of the aorta, pericardium, and mediastinum. *M. Schmidt* has pointed out that there are variations from the normal courses of both nerves.

The vessels of the larynx are derived from the superior and inferior thyroid arteries. The first named gives off: the *superior laryngeal artery*, which reaches the interior of the larynx by the opening in the thyrohyoid membrane, together with the superior laryngeal nerve (see above), sending a small branch along the side of the epiglottis; and the *crico-thyroid artery* (s. laryngea media), which crosses in front of the larynx on the surface of the lig. crico-thyroideum s. conicum, and anastomoses with its fellow artery of the other side, sending, during its course, small branches through the ligament to the inside of the larynx and to the vocal cords.

The *inferior thyroid artery* (of the subclavian) gives off the small laryngea inferior, which ascends, together with the recurrent laryngeal nerve, to the larynx.

The veins show the same arrangement, and anastomose largely with those of other organs, and finally pour their blood into the internal jugular vein.

The mucous membrane of the larynx is the direct continuation of that of the pharyngeal, and, lining the inside of the larynx, forms folds and duplications which have already been described. It is firmly attached to the whole anterior laryngeal wall and to the posterior (laryngeal) surface of the epiglottis, and also to the edge of the vocal cords. On the posterior laryngeal wall, and also on the interarytenoidal part, the submucous tissue is more ample, so that the mucous membrane is movable, and folds on closure of the vocal cords. Also in the ventricular folds, and in particular on the lingual (anterior) surface of the epiglottis and aryepiglottic folds, the submucous tissue is very loose and well developed. This is the reason why we so often find inflammatory or congestive oedema in these situations. Where the mucous membrane is exposed to mechanical frictions, *i. e.*, on both surfaces of the epiglottis, on the aryepiglottic folds, and pars interarytenoidea, and even also on the inner surface of the arytenoid cartilages and vocal cords, it is lined with squamous, at other parts, however, with ciliated, epithelium. In the area where it is covered with squamous epithelium, numerous papillae may be found, which form ridges running sagittally along the vocal cords (*B. Fränkel*). This explains the frequent occurrence of papillary tumours in this region.

Glands are very numerous,—mostly acinous mucous glands,—and they are specially numerous on the laryngeal surface of the epiglottis and its petiolus, on the ventricular folds, and posterior laryngeal wall. On the mucous membrane of the vocal cords they are scanty.

Lymph-vessels of the upper section of the larynx drain into a gland situated between the major horn (cornu majus) of the hyoid bone and the upper margin of the thyroid cartilage; the lymph-vessels of the middle and lower portion drain into the glands situated at the side of the trachea.

The *occurrence of taste-buds* is worth mentioning in the region of the squamous epithelium, viz., on the epiglottis and inside the larynx, on the inner surfaces of the arytenoid cartilages, and on the aryepiglottidean folds (see p. 174), but on the vocal cords there are no taste-buds.

APPENDIX.

The *trachea*, or *windpipe*, is a stiff, elastic tube, passing straight down from the subglottic region of the larynx, but so adapting itself to the curvature of the vertebral column that it sinks more and more backwards until it bifurcates at the level of the fourth dorsal vertebra into the two bronchi. It varies in length from 9 to 12 cm.; its lumen is kept open by fourteen to twenty

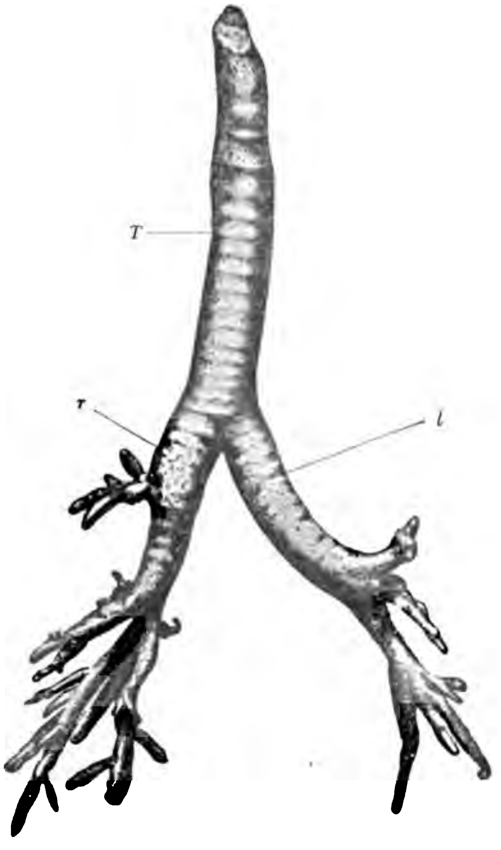


Fig. 100.—Fusible cast of the bronchial tree, frontal view (after *Sahli*): *T*, Trachea; *r*, right bronchus; *l*, left bronchus.

cartilaginous rings, which are connected with each other by tense elastic bands. The diameter measures in children from 5 to 12 mm.; in men, 16 to 22 mm.; in women, 13 to 16 mm.

The cartilaginous rings are not completely closed, but are horseshoe shaped, and form only the anterior and lateral wall of the trachea, while posteriorly the trachea is shut by an elastic fibromuscular wall which is free from cartilages and markedly flattened. The mucous membrane projects somewhat like a spur into the lumen at the site of bifurcation, which projection is called the carina trachea. Of the two bronchi, the right one is wider, shorter, and straighter than the left. This difference is very well shown in the drawing (Fig. 100), which represents a fusible metal preparation by *Sahli,* and explains why foreign bodies much more often reach the right bronchus.

Behind the trachea, the oesophagus passes down, and in the groove between the two structures the recurrent laryngeal nerve ascends (see above). In front of the trachea, between the second and fourth rings, lies the isthmus of the thyroid gland, whilst the lateral lobes of this gland flank the trachea and oesophagus, and further upwards, the thyroid cartilage. From the isthmus a middle lobe sometimes ascends in the mesial line, which must be avoided in superior tracheotomy. (See p. 341.) Many lymphatic glands may be found in the region of the tracheal bifurcation.

The mucous membrane is firmly attached to the trachea, and is rich in acinous mucous glands.

II. PHYSIOLOGY.

The functions of the larynx are those of respiration and production of the voice (phonation).

With regard to respiration, we may consider the larynx as an organ intersecting the air-passage like a tap, in order to regulate the entrance and exit of the air. During quiet respiration the glottis forms a triangular space, which is largest during deep inspiration, when it permits the bifurcation of the trachea to be seen.

Above the entrance to the larynx the air and food paths cross each other; hence there is a possibility of food particles reaching the air-passages. In order to prevent such an occurrence the larynx shuts itself during the act of swallowing. The vocal cords approach each other closely, as also the ventricular folds, the closure of which is made still more secure by the petiolus epiglottidis. It is not yet certain whether the epiglottis plays an active rôle in the closure of the larynx, either by being pushed downwards by the food-bolus gliding over it, or whether it is drawn downwards by the action of the m. aryepiglotticus. Anyhow, the contribution of the epiglottis to the act of closure seems not to be very important, for there are persons who can swallow without any difficulty, although their epiglottis is very rigid, immobile, or even entirely absent. According to *von Bruns*, *Waldeyer*, and *M. Schmidt*, the food-bolus does not glide over the aditus laryngis at all, but the epiglottis is supposed then to be the vehicle, leading the food-bolus along its rolled-in edge into the pyriform sinus, which is admitted to be the passage for fluids.

If, nevertheless, particles of food or foreign bodies are aspirated, then the slightest touching of the very sensitive mucous membrane produces the reflex shutting of the rima glottidis. The increase of the air-pressure in the lung then causes the rima to be explosively opened, which is known as coughing. Not only foreign bodies, which may accidentally have reached the larynx, but also secretion from the lower air-passages, are thus flung out by coughing. Closure of the rima glottidis may also be reflexly provoked by strong contracurrent and irritant vapours. The reflex runs from the superior laryngeal nerve to the medulla, and from there centripetally to the inferior laryngeal nerve.

The larynx plays thus, with respect to respiration, the rôle of a *conductor for the current of respired air*, and also that of a *guard for the lower air-passages.*

With respect to voice production, the larynx may be compared to a double membranous reed-pipe. The reed of the pipe is represented by the two vocal cords, which, in order to produce a sound, must be caused to vibrate by a sufficiently strong expiratory current,* must be closely approximated, and in a certain state of tension. If the two vocal cords are separated for more than 2 mm., hoarseness and even aphonia ensue. The moisture of the vocal cords, which is necessary for satisfactory vibration, is afforded by the mucous glands of *Morgagni's* ventricle, which also affords the necessary room for the vibrations. That it is the vocal cords which produce the sound may be observed from the circumstance that the production of sound is at once lost if the air escapes below the rima glottidis, *e. g.*, after tracheotomy. It may be admitted, however, that by forced approximation of both ventricular folds, as is sometimes seen in nervous patients, at the first laryngoscopic examination, a sound may be produced, but it is only of a noisy, rough, and rattling character. How the closure of the vocal cords is performed has been described in the previous chapter. As to the part which the nasal, oral, and pharyngeal cavities play as resonators, we refer to what we have said before (see pp. 15 and 278).

The human voice is judged by three different qualities: (1) *Vigour;* (2) *timbre;* (3) *pitch* (height or depth).

1. *Vigour* (strength) is directly dependent on the pressure of the expired current of air and the length (amplitude) of the waves of vibration.

2. *Timbre* (shade, nuance), which provides the individual character of voice, is determined by the kind of vibration, this again being defined by the anatomical configuration of the larynx and its resonators. As regards the configuration of the resonator, the voice may appear clear, dull, nasal, or rattling. Age and sex also have a determining influence on the timbre of voice.

3. *Pitch* (height and depth) or register depends—if setting aside the pressure of the expired air—on the number of vibrations, *i. e.*, on the length of the vocal cords and their tension. The highest pitched sounds are produced by the child with its shortest, and the lowest pitched sounds by the adult man with his longest, vocal cords. At the time of puberty the larynx

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* Within certain limits, also, inspiration may produce some noises, or hoarse and cackling sounds, as *e. g.*, in singultus (hiccough).

grows relatively more quickly, and this circumstance, combined perhaps with hyperaemia in young males, causes some kind of physiological disorder of the voice. Anomalies of vibration accordingly occur, and so the impure, rough, "cracked" voice is often produced. This is called "change of voice," or "breaking," which may last for from one to two and even for three years. No change of voice takes place in castrates. As regards register in men, bass, baritone, and tenor, and in women, alto, mezzosoprano, and soprano may be distinguished. Each of these qualifications has its special limitations, which, however, are not sharply defined, and, by training and exercise, may be enlarged more or less towards both extremes of the scale.

Within the various voices different *registers* are distinguishable. The chief registers are the head voice, or falsetto, and the chest voice; between them lies the middle voice, and below them again is the basso or deep voice.

In the *chest voice* the larynx is in its lowest position, and the voice sounds full and strong, and the vibrations can be felt in the chest. The rima glottidis forms a small longitudinal chink; the vocal cords vibrate regularly and to their full extent. In *head voice* the larynx is drawn up, the voice is soft, the resonator reverberates strongly, and the vibrations of the skull may be felt more or less distinctly; only the inner, free margin of the vocal cords vibrates. According to various authors, the rima glottidis, especially in its anterior third, is not so close as in the chest voice. The epiglottis is erected during high-pitched sounds, and sinks back during the low ones; sometimes so much so as to prevent inspection. This, however, is not applicable to all cases. The action of the epiglottis and vocal cords is subject to varying individual peculiarities, which depend on the configuration of the larynx and training of the voice.

In *whispering voice*, or, as one should rather say, in *whispering speech*, the vocal cords are excluded from voice production. The air-current passing through the larynx is used only for voiceless noises, produced by the resonator brought into a suitable and correct position. Whispering, therefore, is possible where voice is lost, *e. g.*, on account of disease of the vocal cords.

Finally, a few words on the central innervation of the larynx may be said. (See Fig. 101.)

We have seen that the larynx serves phonation, which really means the closing of the rima glottidis, and also subserves respiration in such a way that the glottis opens on inspiration.

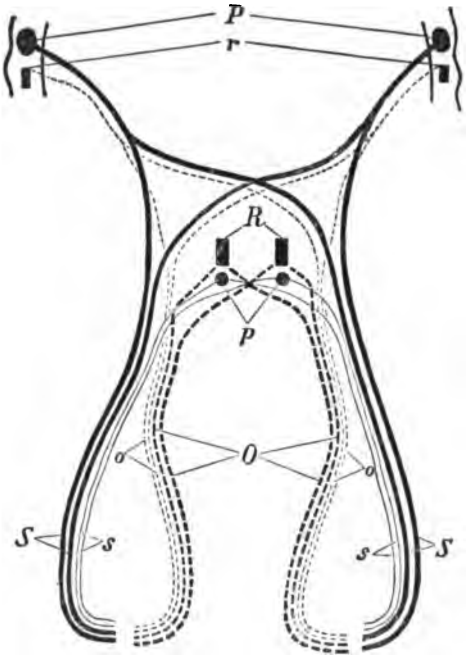

Fig. 101.—Diagrammatic representation of the centers of respiration and phonation in the brain and medulla oblongata and their tracts (after *Réthi*).

The cortical center of phonation (P) is functionally more important, and, therefore, better developed, than the cortical center of respiration, r; on the other hand, the medullary (bulbar) center of respiration, R, is functionally of greater importance than the medullary center of phonation, p. The more important centers are thus marked with capital letters; the minor centers, with small letters; the black non interrupted lines (S, s) show the course of the fibers for the *narrowers*, the lines O, o, those (dotted) for the *dilators* of the glottis.

Both functions have a central representation, and we may at once note that the question of the central innervation of the larynx, which is so important in certain pathological cases, is still in the focus of discussion; nor on account of the difficulties of investigation is it surprising that the opinion of authors on many points is still in opposition.

In quiet, as well as in forced, respiration, according to *Kuttner-Katzenstein*, all the laryngeal muscles, dilators, and narrowers —not only the crico-arytenoideus posticus alone—are innervated simultaneously and constantly; during inspiration the openers get the stronger impulse, while in expiration the narrowers gain in energy.

Quiet respiration, as a pure reflex action, has its center in the floor of the fourth ventricle. This automatic medullary center acts quite independently of the other center situated in the cortex in front of the supra-orbital (inferior frontal) fissure, which rules over *forced respiration*, the latter being a voluntary action. As for respiration, so also for phonation, there exist a medullary and a cortical center. The medullary center, on which we believe that the laryngeal reflex actions depend in certain emotional expressions, such as crying, laughing, coughing, etc., is situated in the fourth ventricle, very close to the automatic center of respiration. Analogous to this we find the cerebral center of voluntary speech and voice very close to the center of voluntary (*i. e.*, forced) respiration.

It is characteristic of the central innervation of the larynx, in respect to respiration and phonation, that both sides are stimulated simultaneously. Therefore paralysis of one center only (on one side) has no influence on the movement of the vocal cords, for the other center at once takes up the action of the other.

The cortical fibers, so it is assumed, run from the cortex to the internal capsule, through the corona radiata, and finally end in the medullary ganglia. Stimulation of these fibers has also a bilateral effect.

III. METHODS OF EXAMINATION.

A. **External examination of the larynx** and neighbouring organs.

Inspection and palpation.

B. **Internal examination.**

1. Testing of function.

2. Inspection.

(*a*) Indirect (laryngoscopia indirecta—shortly, "laryngoscopy").

(*b*) Direct (direct laryngoscopy, autoscopy).

3. Palpation (digital palpation and sounding, probing).

1. TESTING OF FUNCTION.

This must consist of testing of the larynx as to its air permeability, by inspection, palpation, and auscultation, and with regard to the production of voice, by the ear.

2. INTERNAL INSPECTION OF THE LARYNX.

(*a*) **Indirect Method** (*Indirect Laryngoscopy*).—By the indirect method, which is the most important means of examination, we do not get a direct view of the inside, but only an image of it. For this purpose we use a small plane mirror fixed to a long handle, held somewhat obliquely under the uvula, while light is thrown onto it by a reflector (see Fig. 102).

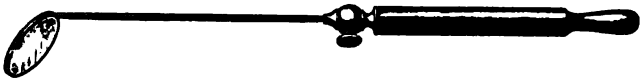

Fig. 102.—Laryngeal mirror.

The source of the light and the reflector have already been discussed in anterior rhinoscopy. (See p. 16.) The laryngeal mirror to be introduced into the mouth is of a round shape, and is fixed to a long handle. Mirrors of several sizes, which can be screwed on to the handle, must be kept handy The larger the mirror, the larger will be the image which we perceive. Small mirrors must be used in examining children or in cases of great stenosis of the isthmus faucium. Disinfection of the mirror is necessary, but is difficult, as the best mirror suffers by being boiled. Under ordinary circumstances it is sufficient to put the mirror for a while into a 5 per cent. carbolic acid solution. Separate mirrors, which should be marked, ought to be set apart for patients with infectious diseases, viz., tuberculosis and syphilis of the larynx.

Technic of Laryngoscopy.—The position of the physician and patient is the same as in rhinoscopy, but patients in bed should be raised up, if possible, or, at least, their heads must be lifted.

The distance of the reflector from the patient's mouth is usually 15 cm., and from the mouth to the uvula, 7 to 8 cm., and from uvula to the glottis, 7 to 8 cm., so that the whole distance from the reflector to the glottis, therefore, will be about 30 cm.; that is, the distance of good near vision in an emmetropic eye.

The patient is directed to open the mouth as wide as he can, for this gives us also the opportunity of inspecting the parts situated in front of the larynx, viz., mouth and throat, and all false teeth should be removed. The patient reclines his head somewhat, and the light is then reflected on to the uvula. While the examiner keeps his head steady, he seizes the patient's tongue, covered with a clean handkerchief, with the left thumb and index-finger, the thumb being uppermost and the index-finger undermost, and pulls it outwards and downwards over the lower jaw. (See Fig. 103.)

It is not good practice to allow the patient to hold their own tongues during the first examination, because they do not hold it firmly enough and permit the tongue to slip backwards. But the patients must subsequently be made to get used to holding their own tongues, in order to allow the physician the free use of both hands.

The laryngeal mirror is held like a pen (both the right and left hands should be well trained), and warmed over a flame with the face downwards, in order to prevent any bedewing by the warm expirated air, and then tested to see if it be too warm. Having convinced himself again that the uvula is well illuminated, the examiner now introduces the mirror face downwards between the tongue and palate, taking care not to touch either, and presses the mirror against the uvula, pushing the latter gently upwards and backwards, at the same time directing the patient to say "eh" or "ehe" while the handle of the mirror is placed in the corner of the mouth (left).

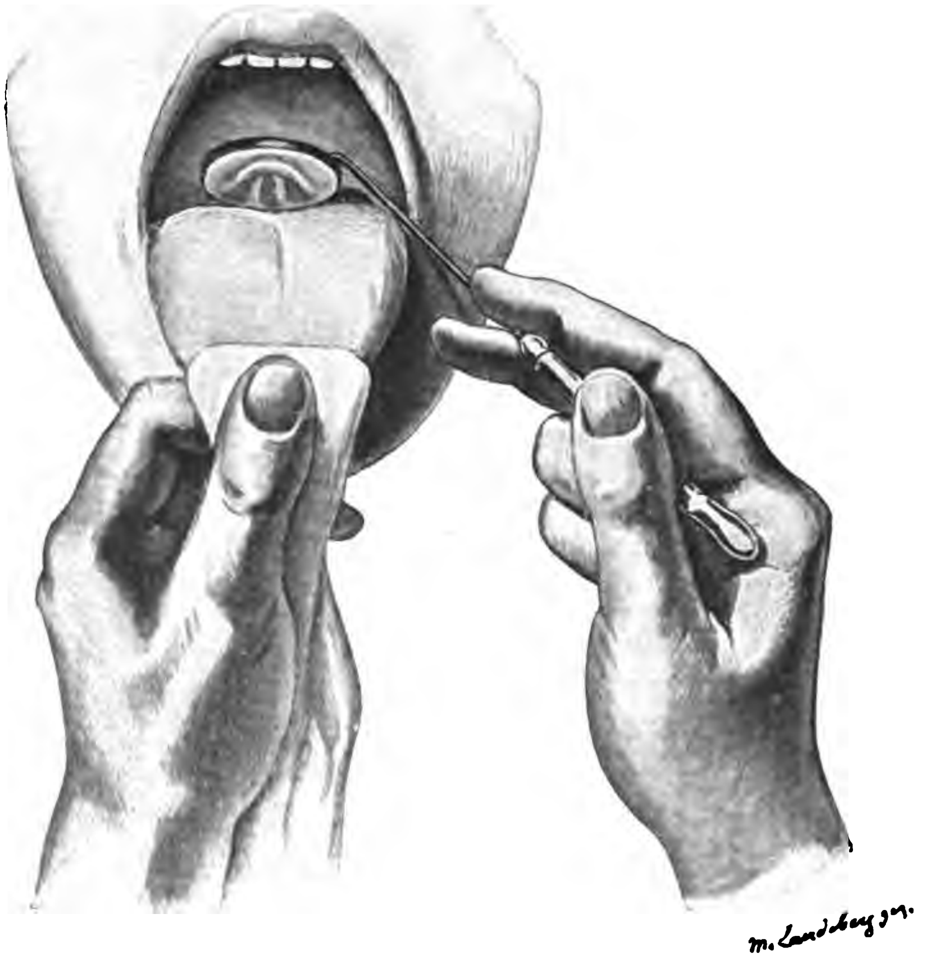

Fig. 103.—Indirect laryngoscopy. Insertion of the mirror (frontal view).

The mirror should be placed against the base of the uvula, and not on its lower end, for the uvula would then easily slip in front of the mirror. After the first introduction of the mirror the patient must be given a rest and ordered to breathe quietly, and it is best then to show him how to do so, as well as how to phonate. Then he is required to again intonate and again to breathe, and so on. It is quite wrong to endeavour to cause him to phonate continuously.

Instead of warming the mirror, which, with electric light and in repeated introduction, is somewhat troublesome, the mirror can be dipped in ½ per cent. lysoform, which, however, does not render disinfection superfluous. After an examination the mirror must be thoroughly cleansed and dried, in order to prevent any mucus from drying thereon.

Difficulties During Examination.—(*a*) *Unskilfulness of the Examiner.*—The beginner usually proceeds too timidly or too roughly. In both cases retching will be the result—in the former, by touching the tongue; in the latter, by the rough handling of the uvula or soft palate. Pulling also too much on the tongue is apt to lead to retching and to the patient interrupting the examination. The examination must be performed with quiet firmness, but yet with a certain gentleness. Practice on the artificial larynx (phantom) is of no real practical value, and it is best to practice on a willing person of slight sensitiveness. If one notices that the light is displaced and does not illuminate the right spot, viz., the uvula, the mirror should be withdrawn and the light correctly adjusted before the examination is repeated. This is far better than to obtain correct illumination by movements of the head, for the light only flickers about and the patient becomes tired and chokes. The same result is to be feared if one holds the mirror too long on the same spot. It is necessary to collect the entire view by slight movements of the handle, and often, undoubtedly, one patient will stand examination better than another, *but it is thoroughly wrong to insist, and to think, that in any case one must finish an examination with the first introduction of the mirror.* Practice makes perfect. It is certainly better to make repeated short introductions in nervous persons, and so to take, as it were, snapshots, which, taken in the aggregate, will give a complete picture.

(*b*) *Faulty Attitude of the Patient.*—Here we have to take into consideration the mental or psychic conditions on the side of the patient; *e. g.*, fear of pain, operation, infection, reflex irritability, which is very often found in nervous persons or alcoholics. These difficulties should be overcome by calming the patient and by explaining to him that which we desire to do, and last, but not least, by scrupulous cleanliness with regard to the mirror. It is a very clever manoeuvre to put the mirror for the first time merely onto the hard palate, and then to pretend to have already seen all quite satisfactorily. Highly nervous patients should be required to breathe abruptly, especially if retching occurs on phonation. Thus the epiglottis is drawn well up at each expiration, and permits of a full view. If choking occurs, the mirror must be at once withdrawn. Local anaesthesia should be used only as an *ultimum refugium*, and for this purpose the base of the tongue and all parts which come in contact with the mirror must be painted with a 10 per cent. cocain or a 10 per cent. alypin solution—a process, however, which does not always overcome the difficulties in question. In some tedious cases one must not lose patience, but must proceed step by step. The tongue is at first pulled out, and the patient then required to repeat in the same manner as before the sound, half spoken, half sung, *e. g.*, "eh," "ehe," so as to get him used to phonation. Then the mirror may be reintroduced. Some patients hold their breath on the introduction of the mirror, and here also the patient has to be trained not to do so. When it is possible, such exercises should be gone through whilst fasting. Many patients involuntarily approximate the lips during an examination more and more, so that they must be reminded to keep their mouths open, although not so wide as to produce a subluxation of the jaw.

Children are difficult to use the laryngoscope on, and in particular, if they have not been trained to permit inspection of the mouth or are badly brought up. If all the arts of friendly persuasion prove in vain, nothing remains than that the child must be firmly held (see p. 112), and laryngoscoped with the tongue depressed, not pulled out. But even thus, one does not always succeed in getting a view, more particularly as the mirror is very often soiled with the mucus brought up.

The best moment to avail one's self of is when the child takes a deep breath whilst crying. Narcosis hardly ever needs be applied; the direct method should rather be resorted to.

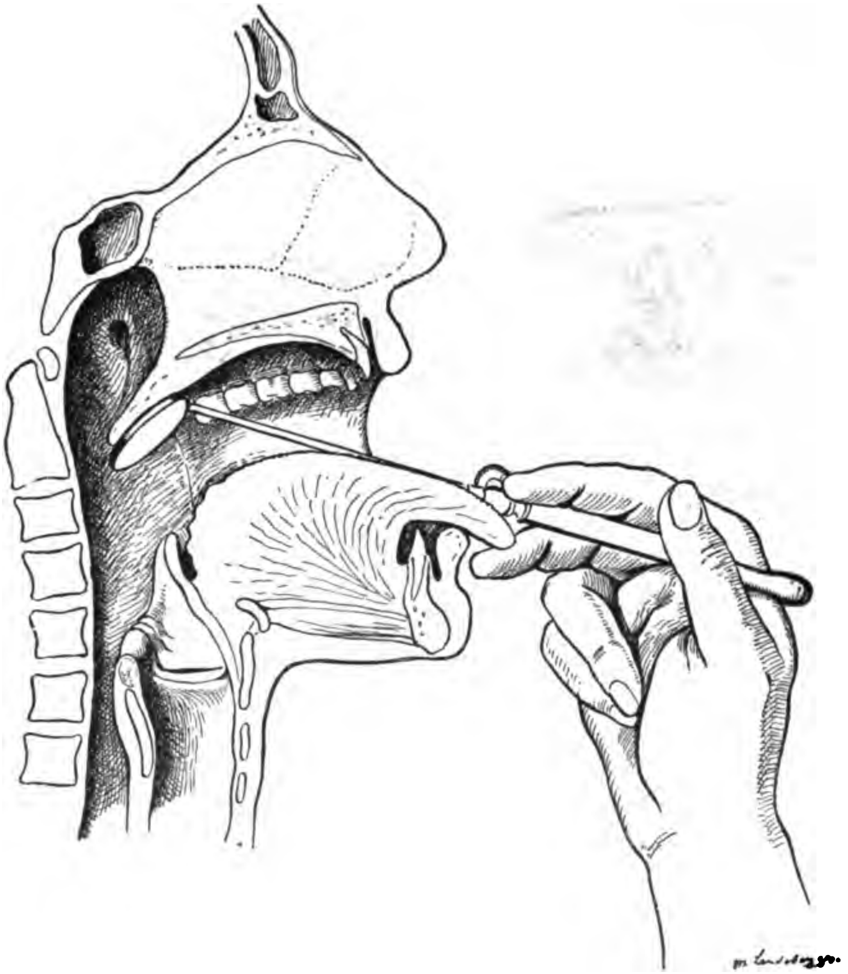

Fig. 104.—Laryngoscopia indirecta. Examination by mirror (lateral view).

(*c*) *Abnormal Anatomical Conditions.*—If the tongue is short, or the frenulum rigid, it is then difficult to grasp the tongue firmly. Here one should take great care not to injure or tear the frenulum by pulling too strongly. This can also be prevented by putting a swab of cotton-wool over the lower incisor teeth.

A too fleshy tongue often rises up so much in the mouth that the mirror cannot be introduced without being soiled. In such a case the tongue must be depressed with a spatula, which requires some force and great skill.

A too *long uvula* easily slips in front of or on to the mirror, if this be too small or has not been placed against the base of the uvula. (See p. 367.) In cases of hypertrophy of the tonsils a small mirror is more suitable, or a preliminary tonsillotomy might be required.

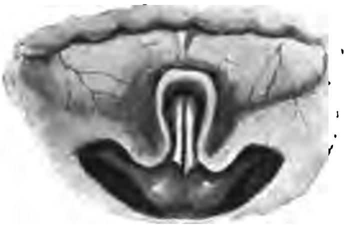

Fig. 105.—Omega-shaped epiglottis.

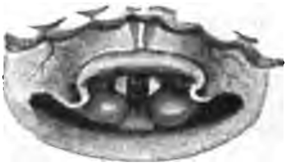

Fig. 106.—Retroflexed epiglottis.

In lordosis of the cervical spine it is necessary, in order to place the mirror sufficiently far backwards and upwards, together with the uvula, to bend the head forwards, or the mirror must be introduced and placed somewhat more laterally.

The epiglottis also often presents a great obstacle: either owing to its being abnormally shaped like an Ω (omega) (see Fig. 105), or it does not raise itself sufficiently (see Fig. 106).

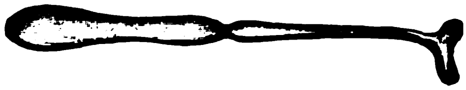

Fig. 107.—Holder of the epiglottis (after *Reichert*).

In general, the higher the phonation, the more the epiglottis rises (see p. 361), and also the more the tongue is pulled forwards. But an exaggerated pulling on the tongue is not permissible, and the height of the intonation also has its limits. The height of intonation which is most suitable for the lifting up of the epiglottis has *to be learned*—not without trouble—*by practice;* and in some cases a high-pitched "A," sung with a head voice resembling an "E," will prove very effectual. In other cases the epiglottis *rises at once* on the mere intention of producing a high "E." At the phonation of "E" the tongue at once goes up. Laryngoscopy succeeds in time if the patient takes several deep, but quick breaths. If one does not succeed by any means, the epiglottis must be lifted by a probe (suitably bent) from the

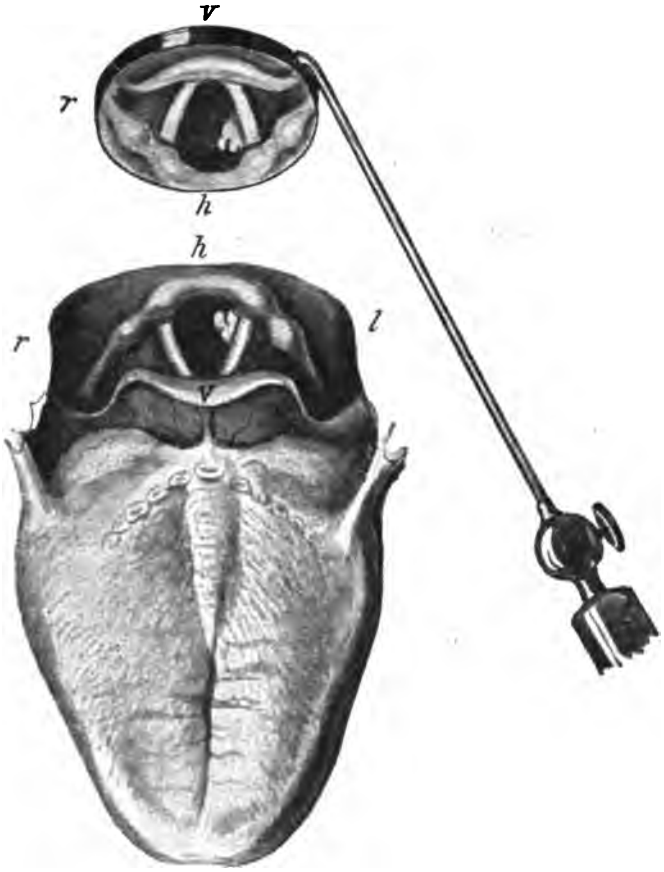

Fig. 108.—Relations of the laryngoscopical image to actuality.

posterior (laryngeal) surface or a *Reichert's* spatula (see Fig. 107) is firmly inserted into the epiglottidean valleculae and the epiglottis anteflected. If the latter be compressed sideways or strongly rolled over, the image of the larynx may be gathered and combined by various positions of the mirror.

The Laryngoscopical Image.—As the mirror, placed against the uvula, is inclined at an angle of 45 degrees towards the horizontal plane, so all that which actually lies in front appears in the upper part of the mirror, and all that actually lies posteriorly, appears at the lower part of the image. But that which is to the right remains also on the right in the mirror, and that on the left remains on the left; it is also termed "left and right," although it appears—viewed from the point of view of the observer—"right and left." (See Fig. 108.).

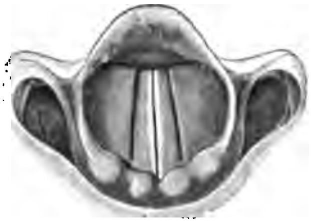

Fig. 109.—Vocal cords during phonation.

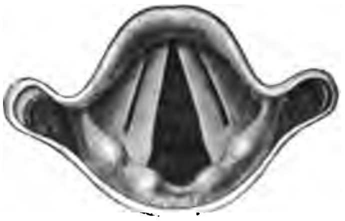

Fig. 110.—The same during respiration.

The several parts—and this the beginner must bear in mind—are defined, as they represent themselves in the patient, not as we see them in the mirror.

When using the laryngoscope the beginner sees, at first, the base of the tongue and the epiglottis, and the glosso-epiglottic ligament stretched out in the middle line, connecting them both, with the valleculae on both sides. Then the posterior (below in the mirror) parts, the arytenoid cartilages, and interarytenoid notch (commissura posterior) will be seen, and lastly—in easy cases even at the first trial—a view of the interior of the larynx is gained, either on respiration or phonation.

The vocal cords are the most conspicuous parts, which approximate closely together on phonation, while on respiration they outline a triangular space, which enlarges on deep inspiration and admits, under favorable conditions, a view still further down into the trachea. (See Figs. 109, 110, 111.)

The vocal processes frequently project somewhat angularly or curvilinearly into the lumen, so that the vocal cords do not always present a straight line. The abduction of the vocal cords is less pronounced during expiration. During forced respiration the vocal cords can sometimes be seen performing short rhythmic opening and closing movements.

Laterally, and a little above the vocal cords, the ventricular folds may be seen, and between both the small chink leading into the sinus of *Morgagni*. *During forced inspiration the vocal cords sometimes disappear under cover of the ventricular folds*, whereby the triangular shape of the glottis assumes a more polygonal, or even circular, outline.

The beginner orientates himself quickest if he searches for the image during phonation, because then the epiglottis offers the least obstruction, and the white vocal cords in the middle of the field cannot easily be overlooked. In some cases, if the endeavour to intonate excites retching, examination during respiration will be preferable. This must be learned by experience.

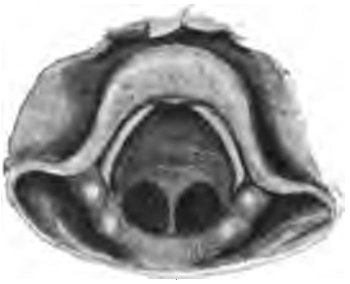

Fig. 111.—Same during deep inspiration.

As a rule, *the larynx should be examined during phonation as well as respiration.*

It is often very difficult to bring the anterior commissure into view, but this is indispensable for the reason that it is the usual site of new-growths. (For the relations of the various parts see Fig. 112.)

It is difficult for the beginner to get, by means of such an image, a correct idea of the accurate distances of the various parts from each other, more especially the distance between the epiglottis and rima glottidis, which is very frequently *underrated.*

Variations of Indirect Laryngoscopy.—Owing to the peculiar form of the larynx, the indirect method is not always sufficient to gain a full view of the larynx, although one rotates the mirror in different directions. Therefore we must resort to some other method or modification which admits of the inspection of special parts not easily seen by the usual method of laryngoscopy.

1. *Inspection of the Posterior Laryngeal Wall.*—The posterior wall of the larynx appears (by usual laryngoscopy) very foreshortened and small; it may be made visible to a large extent down to the bifurcation of the trachea, by *Killian's* method of examination: Whilst the patient is standing and bends the head right down on the chest, the physician, being seated or kneeling, throws the light into his throat from below and places a large mirror *horizontally and a little farther forwards* than usual against the soft palate (see Fig. 114). An electric head-lamp is very useful for this proceeding. This method of examining allows the posterior wall to be seen right down to the bifurcation, and is called *Killian's method of indirect superior tracheoscopy.* (See Figs. 113, 114, 115.) The methods of *Rauchfuss, Rosenberg, and ter Kuile,* which aim at the same purpose, are more complicated. A small mirror on a very long handle is inserted into the glottis itself, so that the image of the posterior wall and of the sublaryngeal (lower) surface of the vocal cords is reflected on a large-

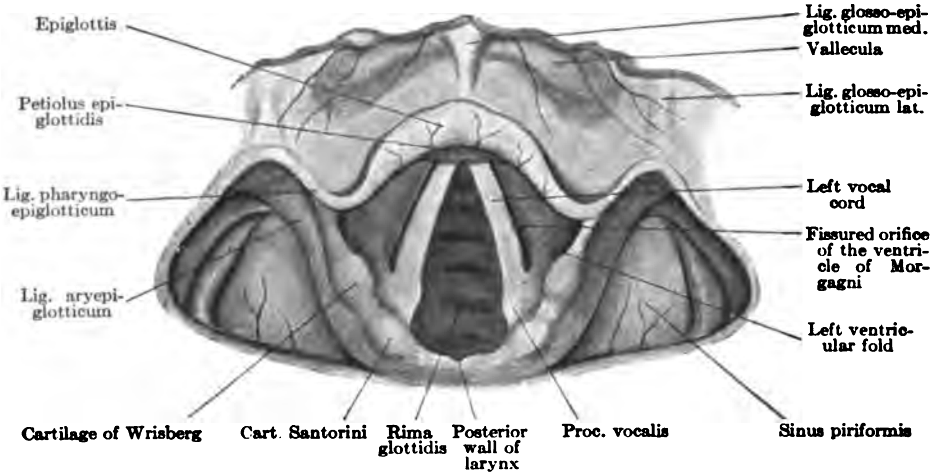

Fig. 112.—Laryngoscopic image (enlarged).

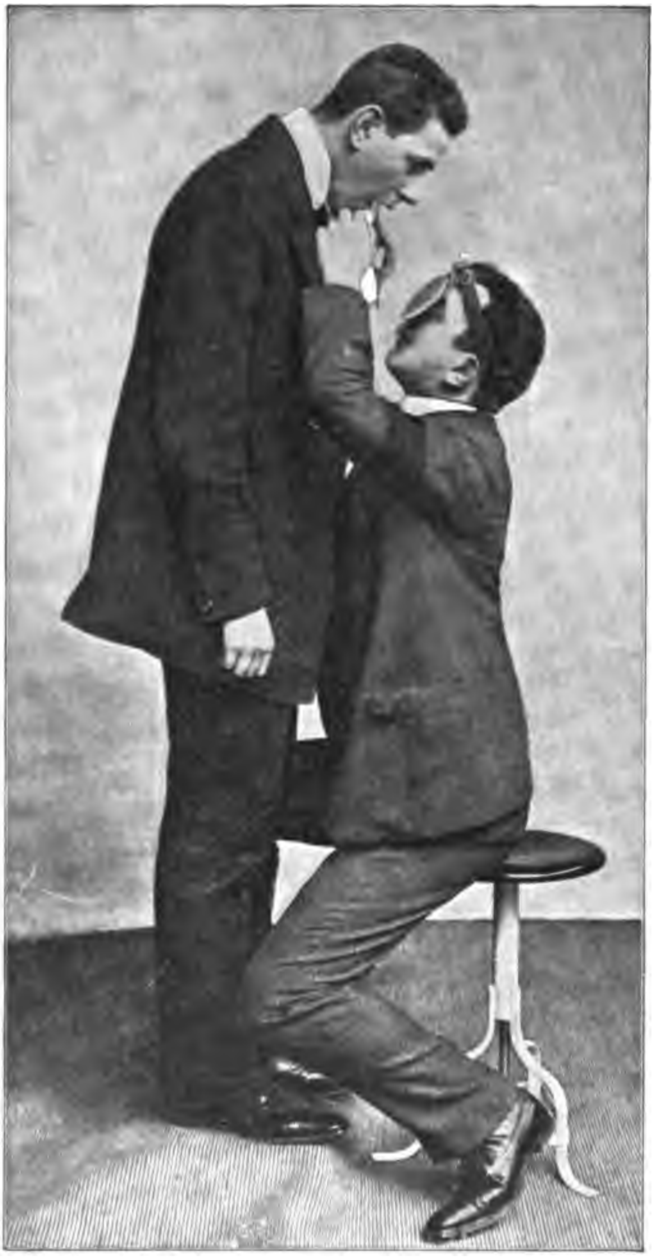

Fig. 113.—Arrangement for the examination by *Killian's* method.

sized ordinary laryngeal mirror placed in the usual position on the uvula. (See Fig. 116.)

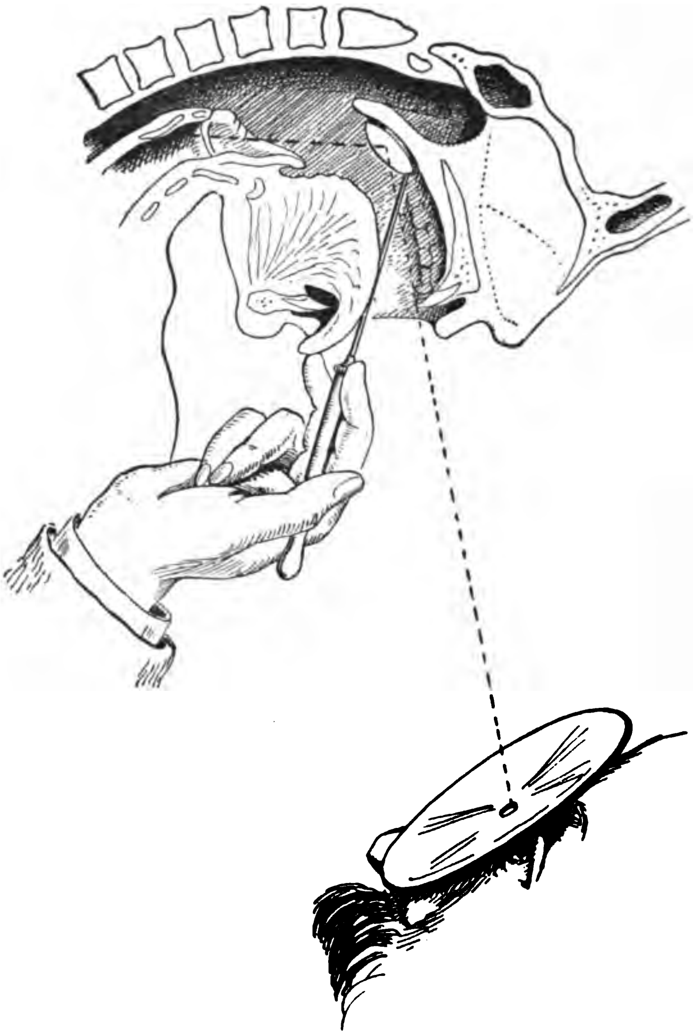

Fig. 114.—Position of the mirror in *Killian's* method of examination.

2. *Inspection of the Lateral Regions* (*Avelli's Method*).—According to *Avelli's* method, the patient is required to bend his head laterally, and a laryngeal mirror is placed at the lower

corner of the throat. By this means the lateral margins of the vocal cords and ventricular folds, as also the ventricles of *Morgagni*, can be inspected. The rima glottidis then appears to be in a slanting position.

3. *Inspection of the Subglottic Region* (*Laryngoscopia Subglottica*).—Under good illumination a large mirror is introduced with the left hand and placed against the uvula, just as in ordinary laryngoscopy. Then, with the other (right hand), a small mirror is inserted into the rima glottidis. The patient is ordered to take a deep breath, and at this moment the mirror is quickly introduced through the opened rima, and the under surface of the vocal cords is inspected by shifting and slightly rotating the mirror in the frontal and sagittal directions. The mirror is of an oval shape, and is fixed on a long and suitably bent handle. For each side a separate mirror is necessary (*Gerber-Magnus*). The aditus laryngis and vocal cords must be anaesthetised. *Gerber* has recently designed a mirror, similar to *Rosenberg's* mirror, which, when in situ, can be unfolded by a special mechanism.

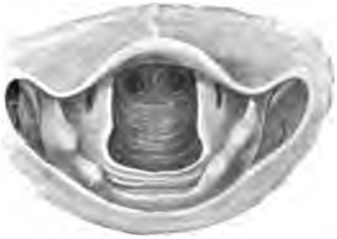

Fig. 115. — Posterior laryngeal wall and bifurcation (*Killian's* method of superior tracheoscopy).

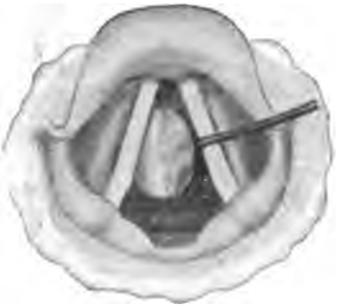

Fig. 116.—Subglottic laryngoscopy.

The subglottic region of the larynx can also be inspected through the wound after tracheotomy by means of a small mirror which is inserted into the wound (*indirect inferior tracheoscopy*). If the mirror, face downwards, is suitably rotated, the whole trachea down to the bifurcation may be inspected. Seen from below, the vocal cords appear not as white bands, but as pinkish or red pads.

(*b*) **Direct Laryngoscopy** (**Autoscopy**).—*Kirstein* was the first to describe this method, which allows the larynx and its various parts to be directly inspected without the aid of a mirror in their natural position. The examiner stands in front of the patient, who bends forwards the upper part of his body, at the same time that the head is bent backwards. A spatula, fixed at right angles to a handle, and bent at its front end (see Fig. 117), is introduced over the root of the tongue, so far as to reach the valleculae, and the tongue depressed firmly downwards and forwards, so that a groove is formed and the epiglottis erected. By this manipulation a channel is shaped, through which the examiner—by means of *Kirstein's* head-lamp—can inspect the inside of the larynx.

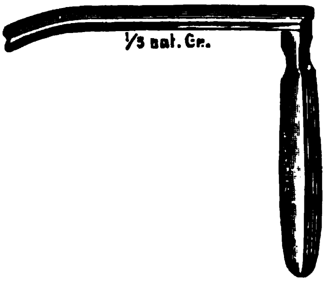

Fig. 117.—Spatula of *Kirstein*.

Autoscopy requires great practice, and is very often felt as an unpleasant experience by the patient, especially if he be very nervous and be possessed of a thick, fleshy tongue. Women and children are easier to autoscope than men. (See Fig. 118.)

Some persons even cannot be examined by *Kirstein's* method. Autoscopy is facilitated by the tubiform spatula of *Killian* (intralaryngeal spatula), which may also be used as a directing staff for bronchoscopic tubes. (See Fig. 119.) Recently the *universal demonstration electroscope of Casper-Killian* has come into use, which carries on the upper end of the handle a small electric lamp, and may be fixed to the intralaryngeal tubiform spatula. (See Fig. 120.)

By direct laryngoscopy the posterior regions and parts of the larynx, and far down into the trachea, may be made visible; the anterior parts, however, are more difficult to inspect.

In any case direct examination of the larynx is a valuable means and supplement to ordinary laryngoscopy, particularly in children, and where intralaryngeal operations have to be performed under general anaesthesia on children.

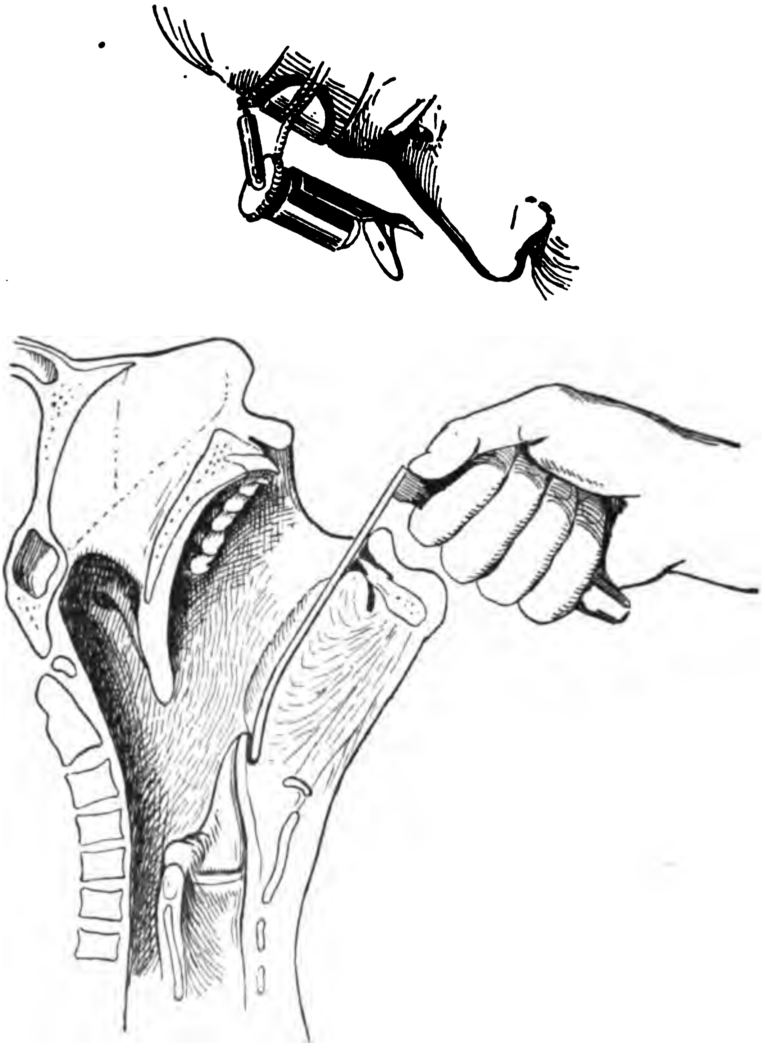

Fig. 118.—Autoscopy (after *Kirstein*) (diagrammatic).

Direct Tracheoscopy and Bronchoscopy.—Following *Kirstein's* idea, *Killian* has described a method which permits the deeper portions of the air-passages, viz., trachea and bronchi, to be directly inspected. The principle of this is to pass a long and strong tube into the trachea or bronchi, and then to throw light through them by means of *Kirstein's* head-lamp or *Casper-Killian's electroscope.* One may distinguish a superior and inferior method of bronchoscopy, viz., if the tube is passed down through the mouth or through a tracheotomy wound.

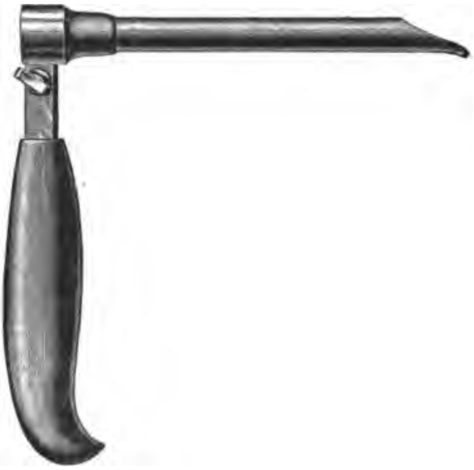

Fig. 119.—Intralaryngeal tubiform spatula (after *Killian*).

Superior, Direct Bronchoscopy.—(*a*) *In Adults.*—Grown-up persons are best examined whilst fasting. They must be seated with the body inclined forwards and the head bent well backwards.

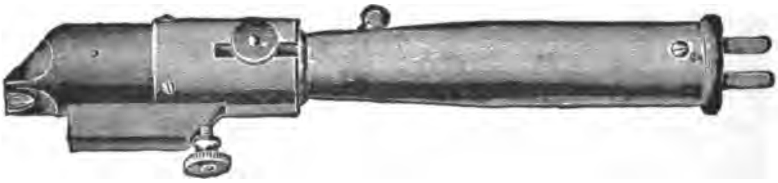

Fig. 120.—Universal demonstration electroscope (after *Casper-Killian*).

The first thing to do is to cocainise the throat, base of the tongue, soft palate, and the larynx, and have the patient seated on a low bench or foot-stool. According to *Killian*, all the parts are first painted with a 10 per cent. watery solution, and then with a 25 per cent. alcoholic solution, to which two drops of adrenalin (1:1000) is added; very excitable persons must be given an injection of morphine about fifteen minutes before the examination.

Then the tubiform spatula (Fig. 119), warmed over a flame, and lubricated with fluid paraffin, is inserted under the guidance of the eye and finger well back, and as far as possible between the epiglottis and posterior pharyngeal wall, and at the moment of deep inspiration is pushed through the opened glottis. The trachea is now cocainised by means of a long straight sponge-holder (see Fig. 121). Through the tubiform spatula a tube, likewise warmed and lubricated, of 7 to 9 mm. in diameter, is now passed down, the length of it varying from 15 to 25 cm. for tracheoscopy, and 30 to 45 cm. for bronchoscopy (see Fig. 122). If one desires to insert the long tube directly, the patient must recline with head hanging downwards.

In order *to reach the bronchi beyond the bifurcation* it is necessary, having cocainised the bronchi, to push them so far towards the middle line until the angle formed by them and the trachea has disappeared. The right bronchus, owing to its straighter course, is easier to examine. Mucus, which might obstruct the view, may be removed by a specially designed syringe. (See Fig. 123.) The bronchoscopic tubes are perforated at the side, so as not to hinder respiration. In superior bronchoscopy general anaesthesia is often required, and here also the recumbent position is necessary.

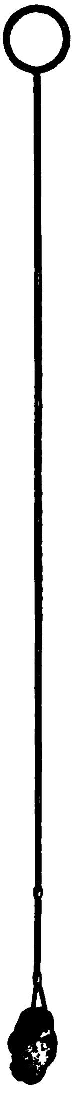

Fig. 121.—Long sponge-holder for local anaesthesia.

(*b*) Children must be examined in the recumbent position, with the head hanging down, and under general anaesthesia; in older children local anaesthesia is often sufficient. Tubes are used of 5 to 6 mm. in diameter, and 10 to 15 cm. in length for the trachea, and 15 to 25 cm. for the bronchi.

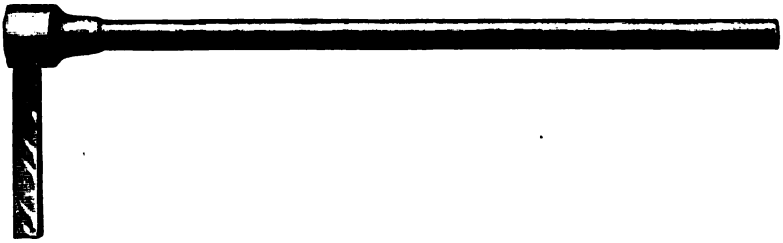

Fig. 122.—Tube for bronchoscopy.

Inferior tracheoscopy and bronchoscopy is much simpler. Narcosis is not necessary, but careful local anaesthesia of the tracheal opening and mucous membrane. The head of the patient is retracted; the chin is turned towards the side, and the tube, having been warmed and lubricated, passed down. The length of the tube may be considerably shorter than for superior tracheoscopy or bronchoscopy.

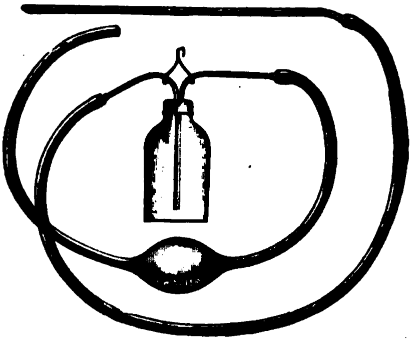

Fig. 123.—*Killian's* syringe for aspiration of the mucus from the trachea or bronchi.

3. PALPATION OF THE INSIDE OF LARYNX.

Palpation with the disinfected finger is of small value and is little used. It is only in children that digital palpation is of any use, on account of their resistance to the mirror and probe.

The finger mostly proves too short in adults, as it reaches only to the epiglottis.

The laryngeal probe, made of flexible metal, is introduced only under the guidance of the mirror. (See Fig. 124.) The handle is seized like a pen-holder, and inserted between tongue and palate as far backwards until the button on the probe appears in the mirror; the handle is now lifted so that the probe is permitted to slip into the larynx. The sounding of the various parts of the larynx under the guidance of the mirror is very difficult for the beginner, owing to the altered perspective in the image. But it should be practised as diligently and assiduously as possible, for skill and dexterity with regard to intralaryngeal manipulations can only be acquired by practising sounding on a willing and locally anaesthetised patient.

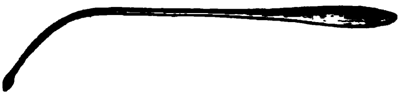

Fig. 124.—Laryngeal probe.

For the purpose of local anaesthesia cocaine is used, and a 10 per cent. solution is sufficient for examination; but for very sensitive persons, a 20 per cent. solution may be used; a 10 per cent. solution of alypin (see p. 22) may also be applied. In certain cases it is useful to combine local anaesthesia with artificial anaemia by means of adrenalin solution 1:1000. The technic of local anaesthesia will be discussed later on.

IV. COURSE OF EXAMINATION.*

ANAMNESIS.

1. *Heredity.*
2. *General conditions of life,* occupation.
3. *Previous diseases:* Previous diseases of the larynx often

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*The proceedings here follow the same lines as in Part I on Rhinoscopy. Owing to the intimate relation of throat, nose, and larynx, repetitions are unavoidable. It is, however, better, not to refer constantly to former descriptions, but to give here a complete account.

leave behind a disposition to further affections. This may also be said of some general diseases, such as influenza, whooping-cough, etc.

4. *Present disease:* Duration, course, origin: cold, occupation or trade injuries (dust, vapours, powdered particles, overstrain of voice, etc.); bad habits (tobacco, alcohol, spices, often combined); medicines (iodine, mercury, arsenic, lead); infectious diseases; diseases of neighbouring organs (nose, pharynx, mouth, thyroid gland, gullet, nerves, etc.) which are liable to affect the larynx; injuries; and *previous treatment.*

5. *Subjective symptoms:* (*a*) *Disorders of Sensibility.*—Complaint of pain is often made, although, on the whole, less often than in affections of the pharynx; viz., pain on speaking, coughing, or even on breathing; oftener on swallowing. Dysphagia usually indicates an affection of the throat; but if nothing can be discovered in the pharynx, one should always also examine the larynx, especially if the patient himself localises his pains more in the lower section of the throat, in the region of the larynx, or in the middle or side of the neck. Accurate localisation is often impossible. The pains are often due to an affection of the aditus laryngis, and radiate towards the ear by the way of the auricular branch of the vagus. Severe dysphagia: *i. e.*, pains which make feeding a torture or impossible, should lead us to inspect the posterior laryngeal wall, which is exceedingly sensitive.

In many cases pain is absent, and the patients complain only of curious sensations (paraesthesiae), such as burning, tickling, soreness, etc. These are mostly due to inflammatory processes or hysteria, and in tuberculosis of the lung also such feelings are often reported. (See Part III, pp. 309 and 331).

(*b*) *Difficulties of swallowing* are often combined with pain during swallowing, already mentioned. Here also the pharynx must be examined first (see p. 245), and if found healthy, then the larynx. But it should be borne in mind that many diseases causing dysphagia (difficulties or pain on swallowing, misswallowing—pp. 246 and 333) may affect the throat and larynx at the same time (diphtheria, bulbar paralysis, etc.). Paralysis of the recurrent nerve is often combined with paralysis of the velum palati.

(c) *Want of Breath.*—Never, or very seldom, can a diagnosis be made as to the site of the cause of dyspnoea from the allegation. In laryngeal dyspnoea *inspiration* is often very difficult.

(d) *General symptoms* are found in phlegmonous processes and in affections of the larynx which follow or are complications of infectious diseases. General symptoms are often very variable, and depend much on the individual sensitiveness and on the extent and seat of the disease.

STATUS PRAESENS.

A. External Examination.—(1) *Inspection: Broadening of the laryngeal region.* In goiter, tumours, injuries, *displacement* of the larynx (marked by the position of the "Adam's apple").

Respiratory Movements.—In laryngeal stenosis the *larynx* conspicuously descends on inspiration, while on expiration the larynx again goes up; in *tracheal* stenosis these movements of the larynx are often *absent,* or only slight.

(2) *Palpation* is a good supplement to inspection, for the movements of the larynx during swallowing and respiration are often better felt than they can be seen. The "stridor" present in stenosis is also often perceived by placing one's finger on the larynx. Examination of consistency and tenderness (very marked laterally in perichondritis of the arytenoid cartilages); crepitation (in fractures), which should not be confounded with the normal grating sensations sometimes felt on movements of the larynx (due to the friction of the superior horns of the thyroid cartilages with the vertebral column).

B. INTERNAL EXAMINATION.

1. Testing the Function.—(a) *Air Permeability.* The air-passage of the larynx and trachea may be narrowed or blocked.

1. *Suddenly,* by the aspiration of foreign bodies, spasms of the vocal cords in tabes, etc.

2. *Within a relatively short time,* in acute inflammations (phlegmon, oedema, or diphtheria).

3. *Gradually,* in chronic affections (tuberculosis, syphilis, scleroma, in bilateral paralysis of the "postici," and compression

of the trachea). (For details of laryngeal and tracheal stenosis see Special Part, p. 408.)

Narrowing of the air-passage results in dyspnoea, which is the more severe the greater the obstruction and the quicker it is developed. In a slow and gradual development, the subjective symptoms are comparatively mild, and in obvious contrast to the objective findings, provided that the patient has remained quiet. The patient usually adapts himself to the gradual reduction of air supply. But any bodily effort, *i. e.*, running up and down stairs, domestic work, etc., at once causes dyspnoea, owing to the greater need for oxygen; and then the dyspnoea may be increased to cyanosis and even occasion asphyxia. The greater the dyspnoea, the more pronounced are the signs: The movements of the larynx are greater and are more easily seen and felt (see above). On inspiration a stridulous noise can be observed (panting, "sawing"), the so-called "stridor," which is due to the friction of the air, and can be felt at the place of origin as a "fremissement cataire." By this sign the site of stenosis may often be determined. In marked dyspnoea respiration is deeper and slower, and is combined with the action of all the auxiliary muscles of the nose, neck, and thorax; the nostrils are blown out at each expiration and drawn in during inspiration, and the jugular fossa, the costal margin, and the ensiform process are all drawn inwards. If the stenosis persists for a long time, the heart becomes involved and shows signs of incompetence; respiration becomes shallower, its rate is increased, and the pulse, at the beginning full and slow, becomes rapid and feeble.

(*b*) *Examination of the Voice.*—The physiological laws of voice production teach us that we must expect an alteration of the voice:

1. If the vocal cords do not vibrate strongly enough, because of the blast of expirated air being too weak, *e. g.*, in general debility; in diseases of the organs of the thorax or abdominal cavity, which are apt to impede or shallow the respiration, in tracheal stenosis, in disorders of mobility of the larynx, etc.

2. If the vocal cords are not approximated closely enough to each other or vibrate in a faulty manner, either from *mechanical hindrances*, *e. g.*, malformations and deformities of the vocal

cords themselves or of neighbouring organs, in particular, of the posterior laryngeal wall; in swelling, tumour, and deposits; or in hindrances of a *paralytic nature, e. g.*, paralysis of the adductors or tensors. Very often several of these causes act in combination.

The alteration of voice may be very slight, but in other cases again is so pronounced and characteristic that an experienced physician is at once enabled to make a diagnosis from it. Thus one speaks of *hoarseness* (paraphony, raucedo), if the voice is accompanied by noises or if it sounds impure, rough, harsh, or obscured; of *aphonia,* if no voice at all can be produced; of *double voice* (diphthonia, diplophonia), if two sounds can be heard simultaneously; of *weakness of the voice* (phonasthenia), if the voice sounds weak, tired, tuneless; of pathological "castrate" voice, or pathological falsetto, if the voice in men frequently changes from the ordinary chest voice to the high-pitched falsetto or head voice, or sounds constantly so.

Apart from tracheotomy, aphonia is met with in lesions of the motor nerves, particularly in hysteria; or in marked anatomical changes.

Double voice (diphthonia) occurs in tumours or more or less in circumscribed thickening or swelling of the vocal lips, which are thus divided into two sections, vibrating differently and independently of each other.

Phonasthenia is frequently caused by severe general disease; but apart from this it points with great probability to a motor lesion (paresis of the recurrent nerve), and in the latter case the voice is weaker, owing to the waste of air during phonation (*Ziemssen*).

While we are occupied with the examination of the voice, we must also notice any coughing which may be present.

Coughing is a defensive reflex action against foreign bodies, accumulated secretions, or other impediments of respiration (see p. 359). In catarrh of the larynx the stimulus to cough, apart from individual sensitiveness, is greater and more frequent, while the coughing itself is more forceful and spasmodic in direct relation to the dryness of the catarrh, viz., the less secretion to be removed. In that sense the cough is spoken of as *dry*, in contrast to a *humid*, moist, or loose cough, whereby masses of secretion are discharged. Dry cough sounds empty, hollow, and rough; a loose cough sounds more full. It is not possible to judge by the sound whether the cough is laryngeal or of deeper origin or is excited by other organs (nose, pharynx, ear, uterus, etc.).

In the larynx it is the posterior wall, the interarytenoid part, which is the most irritable; hence the severe cough in disease of this region. Next in order of importance comes the region of bifurcation of the trachea as being specially irritable. Laryngeal cough "is distinguished by its dryness ('brassiness'), it is more like a scraping to clear the throat." A raucous, loud, bellowing cough points to subglottic changes, and an aphonic cough to excessive swelling of the vocal cords or to bilateral paralysis of the recurrent nerve. In hysteria, coughing remains loud and full, while the voice might be reduced to aphonia. Some patients cough only at night, when in bed and lying down, owing, perhaps, to the collection or shifting of secretion; others chiefly on changes of temperature or on inspiration of vitiated air. "Nervous cough" is very obdurate, and tends to increase if the patient knows himself to be under observation, and then it diminishes if his attention be distracted; during sleep it ceases entirely, though not always.

2. **Internal Inspection.**—The examination must begin with an exact exploration of the *mouth and throat*, and, in particular, of those parts close to the aditus laryngis.

(*a*) *Indirect Laryngoscopy.*—One should not direct the attention exclusively to the vocal cords, a mistake which is committed by all beginners, though they are, by their sinewy white appearance, the most conspicuous parts. The lateral (outer) margins of the vocal cords, on the other hand, which pass towards the ventricle of *Morgagni*, appear, if they are at all visible, of a velvety red.

In smokers, drinkers, and people who speak much, the vocal cords are of a more reddish-grey color, but, on the whole, the color depends on the source of light which is employed; marked pallor, due to anaemia, points to tuberculosis. The margin of the epiglottis often shows well-defined white or yellow spots, corresponding to the cartilage shining through the mucous membrane, or to the mouths of mucous glands, which are often mistaken by the inexperienced for "tubercles."

It has been already pointed out that the vocal cords must be examined during respiration and phonation, so that by this way the motility of these important structures is tested and it is ascertained whether both vocal cords act harmoniously, or whether one lags behind in adduction or abduction, or is altogether immobile, or is fixed in a certain position. Immobility of one vocal cord occurs in disease of the corresponding arytenoid cartilage or its joint, and in paralysis; sometimes also in gross swelling of the posterior laryngeal wall.

The *secretion* must be noticed. Normally only a small amount of mucous secretion is discharged. In pathological conditions the secretion alters. Small masses of mucus may frequently be seen on the vocal cords, or threads of mucus are stretched from one to the other. The secretion may dry up, forming crusts or scabs, which may be seen covering the vocal cords and posterior laryngeal wall, causing hoarseness, pain, and even dyspnoea. In such cases the secretion is mostly derived from the nose or pharynx, and mainly from an atrophic rhinopharyngitis. Acute laryngitis at the beginning also is liable to such a painful exsiccation of its secretion. If haemorrhage is found, the lungs and nose must be examined, as the larynx here plays a secondary part. Small haemorrhages point to erosion or ulceration; an offensive smell, to decomposing or gangrenous processes (carcinoma).

Indirect laryngoscopy is sufficient for the majority of cases. If there are changes on the posterior wall, *Killian's* method will have to be employed. Infiltrations or ulcers on the posterior wall are mostly tubercular in nature.

Only in unique instances will the technically difficult methods of *Rosenberg*, *ter Kuile*, and *Gerber* have to be resorted to, *e. g.*, in subglottic tumours or angiomas, causing haemorrhage, or in disorders of the voice which cannot be satisfactorily explained by what is found above the vocal cords. The number of cases suitable for that kind of examination is very small.

(*b*) *Autoscopy.*—Direct laryngoscopy (see p. 378) is used chiefly in children if examination by mirror is impossible, and also in adults, if a view by the mirror cannot be attained, especially if the jaws cannot be opened wide enough (lockjaw), or on account of retropharyngeal tumours projecting too much and obstructing the view.

Tracheoscopy (see page 379) is indicated in tracheal stenosis (goiter, intratracheal tumour). Tracheoscopy alone permits of a clear idea of the site and degree of the obstruction, and whether an operation is necessary or not. Inspection or palpation from outside is not always satisfactory.

Bronchoscopy (see p. 379) up to now was only used in those cases where, according to the anamnesis and certain other signs, it was assumed that a foreign body had become lodged in one of the bronchi. Before performing bronchoscopy, the *Röntgen* rays should be applied. How far bronchoscopy is useful in other cases is a question for the future.

3. **Palpation of the Interior of the Larynx.**—Palpation, in the form of probing, in order to test the consistency, mobility, and seat of some new-growth, is a useful supplement to inspection; this should be done under local anaesthesia. Tenderness can also be discovered by the probe, but this, of course, without local anaesthesia.

C. OTHER ORGANS.

1. **Nose; Mouth; Throat.**—Diseases of the larynx are often, in just the same way as pharyngeal affections, of a secondary nature. They frequently follow an affection of the mouth or throat, or are continued from the nose or nasopharyngeal space. We must bear in mind this connection, and combine the laryngoscopic examination with an inspection of the mouth and throat (see p. 364). Frequently the affection of the larynx and trachea only forms a part of a descending catarrh of the upper air-passages.

2. **Lungs.**—The relationship between larynx and lung appears in tuberculosis. The conditions of the lungs in many cases gives us the first indication of the true nature of the laryngeal affection. In haemorrhages one should never omit an examination of the lungs. Larger quantities of blood, which, for instance,

exceed a teaspoonful, are seldom derived from the larynx itself, but mostly from the lung or nose or nasopharynx.

3. **Central Nervous System.**—Nervous disorders, sensory as well as motor lesions, are very often due to cerebral, medullary, or spinal disease. Hysteria plays a great rôle in laryngeal affections.

Besides the central nervous system, various other organs also in the region of the neck and chest, owing to the large area supplied by the vagus, are sometimes the cause of nervous disorders in the larynx. In such a case one must discover it by an examination of the entire body.

4. **Reflex Disorders.**—The larynx, like the nose, is also apt to excite reflex neurosis and disorders in other organs, although rarely.

V. TREATMENT.

1. GENERAL MEASURES.

In laryngeal and tracheal affections, as in those of the nose and throat, general treatment is of great importance, often rendering local treatment superfluous, if, indeed, as in the first stage of catarrh, local treatment be not really harmful. In many cases it suffices, and this is markedly so in affections of the larynx and trachea, to exclude every cause of irritation,—change of temperature, alcohol, tobacco, etc.,—in order to procure restoration to health or at least great improvement. We must here emphasise again the need of care, so as not to be misled into overdoing local treatment in the too circumscribed purview of the specialist, or else the same fate will overtake one, as it did that laryngologist who once, to a circle of his colleagues, showed with proud self-satisfaction the larynx of a phthisical patient which he had treated by caustics and curette, and concluded his demonstration with the words, "the *larynx is cured; but—the patient died.*"

We do not mean to say anything against reasonable local treatment in a given case; certainly let it be "est modus in rebus." It is just the rhinologist and laryngologist who should resist the temptation of an instrumental technic, which is growing so vastly just now in the field of their specialty. One must never lose sight of the connection with general medicine.

Among the general measures to be taken in diseases of the larynx and trachea, especially in catarrhal affections, *balneotherapy* must be mentioned first. This is almost traditional. Climatic and hydrotherapeutic and mineral water treatment are the rule. The same principles as in the treatment of pharyngeal disease are here available, but we should like again to point out that all these cures have mostly the effect of only changing and bettering the general conditions of life, and so act indirectly on the easily influenced larynx.

With regard also to diet, we must refer to previous chapters. The larynx comes so much in contact with the food that regulation of the diet, just as in affections of the pharynx, is necessary. The protection of the larynx here stands before everything, and this is applicable not only to external, but also to internal, causes of disorder. Sparing the voice, therefore, is the most important, and is only contra-indicated in functional disturbances arising from an hysterical basis.

A word on the taking of medicines. Sedatives and narcotics may be used against cough, pain, and spasm; expectorants, to facilitate or loosen secretion, and also emetics and purgatives and sudorifics. Opinions are divided as to the usefulness of "solvent" and "irritative" expectorants. I, for my part, do not value them much, but somehow or other one cannot do without them, and one may prescribe them in combination with morphine, codeine, and aqua laurocerasi, etc.; and consequently we must always bear in mind that it is the narcotic substance which abates inflammation by reducing irritation and coughing, and thus indirectly diminishes secretion. A really useful solvent is potassium iodide, which, in many cases, affords relief. It must, however, be cautiously given, as it disagrees with many people. It may be given (3–4 in 200 of water), one teaspoonful every two hours to adults.

The following are useful prescriptions:

℞.	Inf. rad. ipecac	0.5 : 170.0
	Liq. ammon. anisat	5.0
	Morph. muriat	0.03–0.05
	Syr. alth. ad	200.0

F. M. Sig.—One tablespoonful every two hours.

℞. Decoct. rad. senegae........................ 10.00 : 175.00
Liq. ammon. anis.
Aq. amygd. amar........................āā 5.0
Syr. simpl. ....ad 200.0
F. M. Sig.—One tablespoonful every two hours.

℞. Ammon. chlorid............................5.0
Succi. liquirit. (glycyrrhizae)................2.0
Aq. destill. ....ad 200.0
Mixtura solvens. (To this can be added 0.03–0.05 morph. or 0.2 codein. phosphat.)
F. M. Sig.—One tablespoonful every two hours.

℞. Liq. ammon. anis..........................5.0
Aq. amygd. amar..........................10.0
F. M. Sig.—Fifteen drops in a glass of warm sugar water, thrice daily.

℞. Morph. muriat.............................0.15
Aq. amygd. amar...........................15.0
F. M. Sig.—Fifteen drops thrice daily.

℞. Codein. phosph............................0.3
Tinct. bellad..............................5.0
Aq. amygd. amar...........................15.0
F. M. Sig.—Twenty drops thrice daily.

℞. Pot. iod..................................3.0–4.0
Aq. dest..................................200
F. M. Sig.—One tablespoonful every two hours.

℞. Inf. rad. ipecac........................... 0.5 : 180.0
Pot. iodid................................3.0
Syr. simpl. ad............................200
F. M. Sig.—One tablespoonful every two hours. (*Eichhorst*)

In feverish patients who suffer at the same time from indigestion:

℞. Apomorph. mur............................0.05
Morph. mur...............................0.03
Acid. mur................................0.5
Syr. simpl...............................20.0
Aq. destill.............................ad 200
F. M. In vitro flavo.
Sig.—One tablespoonful every two hours.

℞. Sol. acid. phosphor. dil......................5.0 : 180.0
Codein. phosphat..........................0.3
Syr. rub. idaei (raspberry) syrup*...........20.0
F. M. Sig.—One tablespoonful three times daily. (*Eichhorst.*)

For children:

℞. Inf. rad. ipecac............................0.15–0.3 : 100.0
Liq. ammon. anis..........................0.5–3.0
Syr. simpl................................20.0
Sig.—One dessertspoonful every two hours.

℞. Decoct. rad. senegae........................ 5.0 : 100.0
Liq. ammon. anisat.........................0.5–1.5
Syr. alth. ad..............................120.0
Sig.—One dessertspoonful every two hours.

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* Analogous to syrupus mori (wild raspberry).—*Translator's Note.*

℞. Sol. pot. iod................................1.5–2.0 : 100.0
Aq. menth. pip..............................20.0
Sig.—One dessertspoonful four times daily.

℞. Codein. phosphat............................0.01–0.02
Aq. destill..............................ad 50.0
F. M. Sig.—One teaspoonful in sweetened water four times daily.

2. LOCAL TREATMENT.

(a) **Fomentations.**—The technic and indications for cold, warm, and hot fomentations have been described in Part III, p. 250, et seq. What has been said there with regard to diseases of the pharynx is pertinent, ceteris paribus, to affections of the larynx.

(b) **Light Treatment.**—The use of sunlight, especially certain kinds of rays, for treatment is still in the tentative stage, and in regard to the larynx, it remains with the future whether it will be used to any greater extent. Up to the present, reflected sunlight has been used in the treatment of laryngeal tuberculosis (*Sorgo, Kunwald*). The *Röntgen* rays also have been tried in tuberculosis and malignant tumours of the larynx.

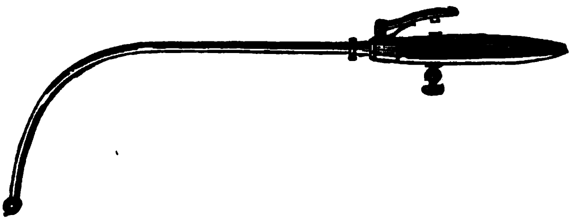

Fig. 125.—Laryngeal electrode.

(c) **Electricity.**—The faradic and, more seldom, the galvanic current is applied either in the milder form extralaryngeally, or, for stronger action, intralaryngeally. In the former the electrodes are usually placed one on either side of the thyroid cartilage. (See Fig. 125.)

In the intralaryngeal application the one (plate) electrode is placed outside on the larynx, or on the sternum, and the other (button) electrode, which must have an arrangement to interrupt the current, is introduced under *guidance of the mirror*, and with *local anaesthesia*, into the larynx. The current is made to act after the button of the intralaryngeal electrode has been brought into the exact position desired. At first the treatment must only last for a few seconds, but later on the sitting may be extended in duration.

(*d*) *Treatment by Drugs.*—Medicaments are introduced into the larynx, in either a fluid state, by painting, or instillation, or in the solid form by insufflation, and for cauterisation; or, again, atomised or vapourised by sprays or by inhalation. (See Fig. 126.)

The larynx is painted under the guidance of the mirror, and very nervous patients ought to be treated—at least at the beginning—by having the larynx previously cocainised. Painting, which is done without the aid of the mirror or by the patient himself, is nothing but useless torture; the brush probably never enters the larynx, which may be considered, with due respect to the crudeness of such manipulation, a rather lucky circumstance. Owing to the difficulties of cleaning a brush, it is better to use in its place small swabs of cotton-wool which are soaked with the medicament and fixed on a holder. (See Fig. 126.) Great care must be taken in introducing the sponge-holder so as not to touch the tongue with the unpalatable fluid. On seeing the swab in the image, the handle of the holder must be lifted, and while the patient phonates, the swab is quickly inserted into the larynx and brought into contact with the part to be painted (see Fig. 127).

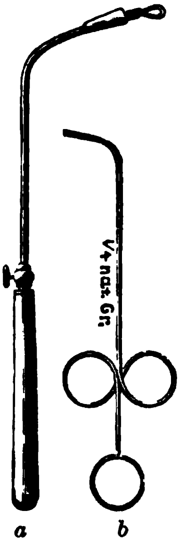

Fig. 126.—Sponge-holder for the larynx: *a*, After *Heryng*; *b*, after *Hartmann*.

In order to augment the effect, the instrument may be moved to and fro, and so the fluid is thus brought into more intimate contact with the parts concerned, and I venture to say that the massage exercised by this latter modus operandi has also in itself a beneficial influence.

Laker approves very much of the therapeutic value of this intralaryngeal massage, particularly in cases of chronic catarrh.

The value of painting the larynx is certainly often exaggerated, and it is frequently continued for a long period, although "nothing particular" or perhaps "only a slight discolouration" of the vocal cords can be found. In these cases the larynx should be painted very moderately. Chronic catarrh and ulceration form the chief indication for painting and brushing.

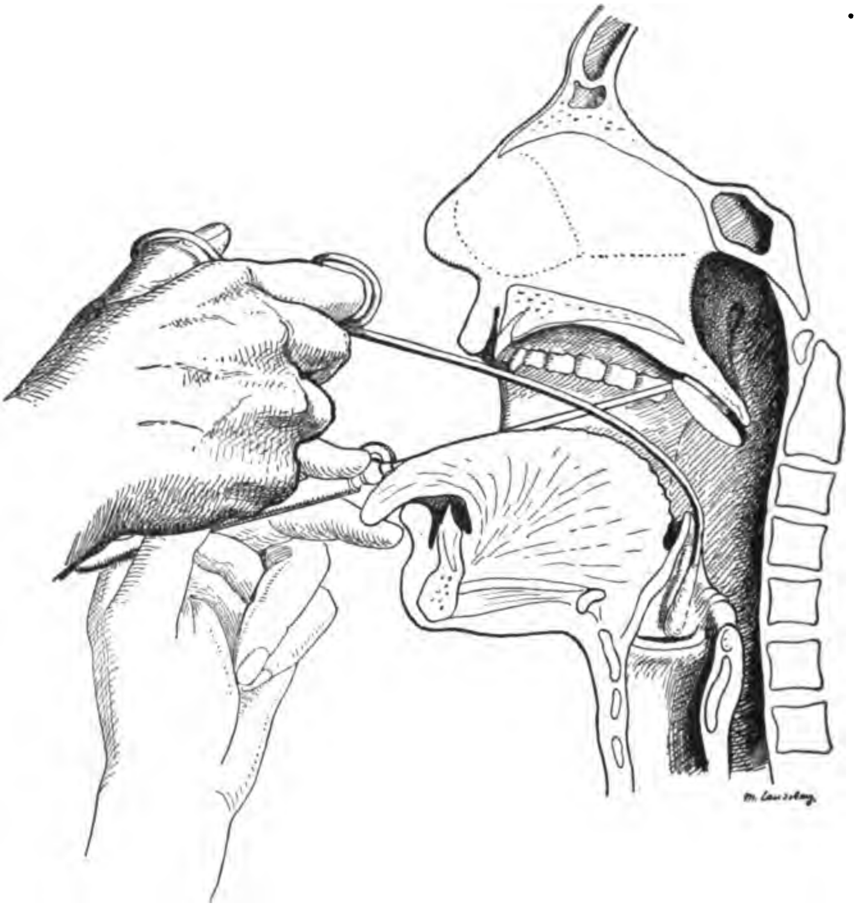

Fig. 127.—Painting the larynx. Introduction of the sponge-holder under guidance of the mirror (diagrammatic).

Instead of the sponge-holder, *a syringe may* be used, by means of which the solution is instilled during phonation. This kind of application is preferred in cases where one desires to instil the medicament drop by drop (*e. g.*, for local anaesthesia), or in order to inject a larger quantity of fluid at once, *e. g.*, menthol in tubercular ulcers, or tepid water for the softening of crusts. (See Fig. 128.)

Insufflation is done with a suitably bent insufflator; but I do not think that this can replace painting, for it does not allow, as has been already pointed out (see p. 253), an exact localisation of the application. I only use insufflation of the larynx in cases of widely spread ulceration.* (See Fig. 129.)

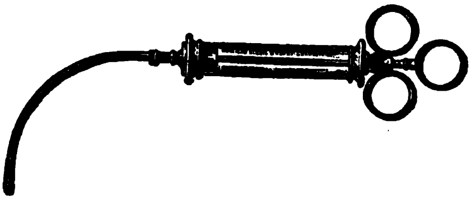

Fig. 128.—Laryngeal syringe.

The larynx, as is also the nose, *may be cauterised* by silver nitrate, chromic or trichloracetic acid, under local anaesthesia, and guidance of the mirror, by means of special cauterisers.

Complications Following Local Methods.—The larynx reacts to all these intralaryngeal manipulations in more or less violent manner, *e. g.*, by cough, sometimes so aggravated as to cause vomiting; by severe irritation, a sense of scratching and burning in the larynx, and spasm of the glottis. Coughing means that the medicament has really come into contact with the larynx. If, however, the patient coughs very much or complains of a sensation of burning, he must be ordered to take some peppermint tablets or menthol pastils. Spasm of the glottis is some-

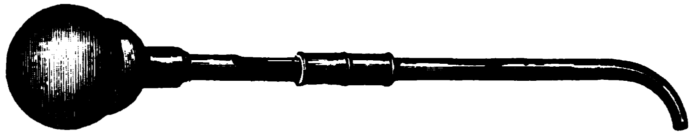

Fig. 129.—Laryngeal insufflator.

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* See also foot-note, p. 253.

what alarming, but not dangerous; the chief thing to bear in mind is *that the physician should not lose his head;* he should calm the patient, and smack him on the back, pat his shoulders, etc.; and if the attack does not quickly pass away, he should compress the patient's nostrils while the latter is made to take several deep breaths. In other cases the patient should be requested to stop breathing as long as he can, or to take a mouthful of water. As a rule, all these complications occur only in very nervous patients on the first occasion of treatment; and consequently, previous anaesthesia of the mucous membrane is to be strongly recommended. *The larynx later on* becomes very tolerant of all manipulations, and does not any longer react even by coughing. The patient is advised to keep silent for about a quarter of an hour after the painting, or he is directed to speak only in a whisper.

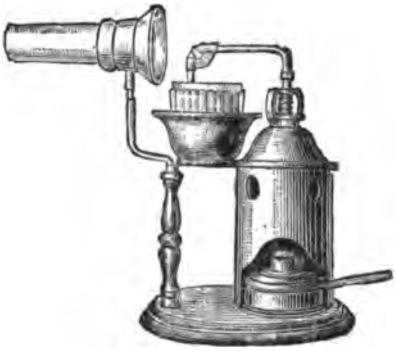

Fig. 130.—Apparatus for inhalation.

Inhalation—(*a*) *of atomized fluids* is done by means of special sprays worked by two bulbs; but, better still, by one of the numerous steam inhalers, which make steam the vehicle of the fluid (atomised) medicament. The inhalatoria existing in so many watering-places and health-resorts work on this same principle. There are many apparatus constructed for inhalation by *Bulling, Wassmuth, Heryng, Nicolai,* etc., each of which is said to have special advantages. When inhaling, the patient should bend his head back and stretch his tongue out, but, nevertheless, the greater part of the vapourised fluid will not reach the larynx at all, but will be retained on the mucous membrane of the pharynx. If it reaches the larynx, coughing will be excited. After the inhalation the patient should remain in the room for a short while.

(*b*) *Of gases*, is generally easier and more effective, because the mucous membrane of the air-passages is brought into intimate contact with the inspired air (gas). The medicaments are volatilised (vapourised) by proper instruments (*Sänger's*, *Rosenberg's*, etc., vapouriser).

The benefit accruing from inhalation must not be overrated, more especially the inhalation of atomised medicated fluids. Their chief effect is due to the heated vapours. It is, therefore, quite superfluous to prescribe all possible sorts of substances for the purpose of inhalation, but one should endeavour to assist the solvent action of the steam. If one desires to stimulate secretion or to loosen the scabs and thus afford a more free respiration, a little salt of Ems or common salt may be added to the water in the inhalation apparatus; or a 1 to 2 per cent. solution of bicarbonate of soda or borax or tar-water (aq. picis and pure water part. aeq.) may be ordered. The effect of inhalation of menthol and thymol is astringent, and, at the same time, slightly disinfectant and deodourant. Peruvian balsam, oil of turpentine, and eucalyptus, etc., and lately inhalations of lignosulphite, have been much recommended; and the vapours of menthol have also a decidedly analgesic effect.

The general practitioner, who has no inhalatoria at his disposal, will satisfy himself with ordering an inhalation apparatus for the purpose of atomising, or in special cases, where the deeper air-passages are effective, a volatiliser. Among the poorer classes a jug filled with boiling water, to which some useful drug (turpentine, etc.) is added and covered with a funnel, will be found efficient. One may also improvise a useful inhaler by means of a kettle, which, being half filled with water, is heated over the fire-grate or a spirit-lamp, and then a long paper tube can be adjusted over the spout.

(*e*) *Operative Treatment.*—(1) *Local Anaesthesia.*—For the local anaesthesia of the larynx, just as for the nose, cocaine or alypin in 10 to 20 per cent. solution is useful; the weaker solution for the slighter manipulations, such as painting, cauterisation, etc.; the stronger solution for more extensive operations; and in certain cases, even a 30 per cent. solution is sometimes necessary. To increase the effect of the cocaine, and also in order to economise the drug, it is well to anaemise the parts concerned prior to the application of the cocaine by means of adrenalin solution

(1 : 1000). I am accustomed to instil a few drops of both drugs into the larynx by means of a syringe and, after a while, to test the mucous membrane with a laryngeal probe as to its sensibility. The effect of the drug on the patient is a feeling of numbness or "a lump in the throat," which, however, does not trouble him so much as the inability to swallow.* If, after five minutes, the anaesthesia is not complete, the instillation is to be repeated, if necessary, several times. If one desires to avoid all unpleasant incidents, it is good practice also to render the epiglottis and the pharynx (velum palati, tonsils, posterior wall) insensitive. Insensitiveness of the mucous membrane usually lasts for five to seven minutes, and considerably longer if adrenalin has been used.

Alypin is less poisonous than cocaine and does not contract the blood-vessels. It is, therefore, very useful if one desires to remove small tumours which are difficult to attack if shrunken, and in this event adrenalin will also be undesirable.

Submucous injections are superfluous.

Poisoning by cocaine seems to be more frequent in laryngeal applications than in the nasal ones, and, according to *Schech*, the poisoning is due to some portion of the solution—in spite of all precaution—having been swallowed and absorbed by the digestive organs. I have never seen poisoning by alypin. For the symptoms of cocaine poisoning see p. 22.

(2) *General anaesthesia* is only induced in exceptional cases, where local anaesthesia is not sufficient, *e. g.*, in children. (See p. 42.)

3. INSTRUMENTS.

Many instruments are used in laryngological practice, and comprise, apart from the galvano-cautery and other electrolytic instruments, various kinds of knives, curettes, forceps, punch forceps, snares, etc., all of which are fixed on suitably bent handles and ought to be as slender as permissible, so as not to hinder the view of the larynx.

With regard to galvano-cauterisation and electrolysis, the same is here pertinent that has been said in the analogous chapters on the nose. (See p. 42, et seq.) As a rule, one does not often use the galvano-cautery in the larynx, if for no other reason than for the great reaction it causes; and that electrolysis is so difficult to apply to the larynx, and its effect so slow, that it is only resorted to very occasionally. (See Figs. 131 and 132.)

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* To be on the safe side, and in order not to be alarmed by the patient, one should point out to him that such and such a discomfort will occur but will pass away.

Galvano-cauterisation and electrolysis are chiefly indicated in cases with vascular tumours; telangiectases (naevi) also readily yield to the caustophor. *Heryng* recommends electrolysis for the purpose of destroying hard, diffuse, tumour-like infiltrations of a tubercular nature.

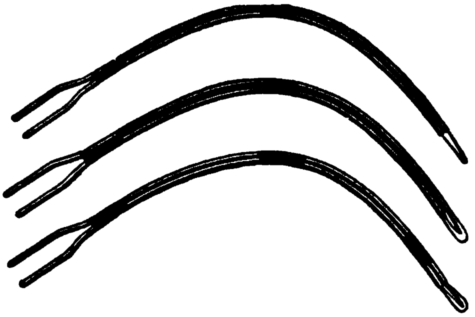

Fig. 131.—Galvano-caustic burner for larynx.

The particular instruments required will be described in the special part. Here we desire only to impress some important points in regard to intralaryngeal surgery.

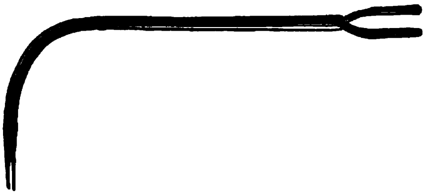

Fig. 132.—Double needle for electrolysis.

The patient must be "trained" before every operation. He must become used to permit the introduction of a large mirror, and also if this be done with the operator's left hand; he must learn to hold his tongue himself, and to breathe quietly and deeply and phonate properly. He must get used to the intralaryngeal probe—eventually with the aid of cocaine, etc.

The instruments should be sterilised in the ordinary manner, and warmed before introduction. The latter must be done always under guidance of the mirror, some instruments used for dilatation being excepted. One should always make sure that the instruments "work" properly, that they are safely fixed to their handles, so that they will not refuse to work, or slip away at the critical psychological moment.

VI. HYGIENE AND PROPHYLAXIS.

In the previous three Parts, which treat of the diseases of the nasal, oral, and pharyngeal cavities, a whole series of important points referring to hygiene and prophylaxis have been discussed, which, directly or indirectly, are also applicable to the larynx. This can easily be understood, considering the intimate relations mentioned on various occasions existing between the several portions of the air-passages, and it suffices only to refer the reader to the previous chapters (pp. 44 and 254). Here we desire only to say something concerning matters which are necessarily connected with the larynx as the organ of voice production.

It is quite remarkable how little the voice is cared for generally. If there is such a thing as "vocal hygiene," it is mostly and chiefly for the appreciation of its artistic value, and may be also that the actor is allowed to share the benefits of rational voice production. That the voice should be carefully watched and protected from childhood, only few will believe; and many physicians even neglect this very important question. Parents think it right, from the pedagogic point of view, if their offspring yells as much as he likes. "Yelling is healthy and expands the chest" is the usual saying. This is, however, true only to a certain extent, and the physician should not miss the opportunity of making it clear to the parents and all concerned that, owing to the great delicacy of the youthful larynx, any continuous strain of the voice during tender years produces damage lasting for a long period, if not forever. This is applicable not only to infants, but still more to older children. Many children fall into the habit of shouting on every occasion and of yelling while under the excitement of playing. They love to imitate the voices of animals, and so overstrain their own voice, and when they have to attend school, do so with an already injured vocal organ. I myself remember several children with nodular or diffuse thickening of the vocal cords who had obviously maltreated their voices by overloud speaking and singing, without their parents thinking it necessary to restrain them. The evil is aggravated when, later on, in school, the children with such irritable and spoiled voices have to attend singing. Under normal conditions there is no objection to singing exercises in the schools, for they are, so to say, a form of health gymnastics with the object of expanding the lungs. But I consider it wrong to compel little children, and, still earlier, at home, in the kindergarten, or "infants' schools," to sing exercises, for there is absolutely no trace of a correct utilisation of the natural means, for all exercises are only play, and the child should sing for its own enjoyment. In my opinion the question whether children should attend singing exercises or not before they go to school must be decided in the negative.

The practitioner's attitude towards singing in school is different, provided that the vocal organs of the children are healthy. Unfortunately, voice production in schools often leaves much to be desired, and it is particularly so with singing in chorus, which causes a great strain, which some children are not able to bear with due regard to their vocal organs.

If the physician notices signs of vocal fatigue or other disorders, or if his attention is directed to this circumstance by the parents, then he should not hesitate to prohibit the child from the regular attendance at singing, temporarily or permanently, as the case may be.

During the time of mutation ("breaking of the voice") singing should be entirely abstained from. To refrain, however, from speaking, is not only unnecessary, but it is useless, provided that no real signs of irritation, in any degree, are apparent, as may easily happen. Shouting, of course, should be forbidden, and if one is asked for advice, the *adoption of the low register* *is to be recommended.* Special treatment is unnecessary. Making fun of or laughing at "mutating" boys or girls has bad consequences, because they then try to correct what they believe to be their own fault, and thereby more and more strain their voice, and so are likely to spoil it for all time. They must be relieved from the obligation of school singing, and not be permitted to sing until the change of voice is completely finished and a definite voice (register) has developed.

In later years the advice of the physician is not much sought with regard to the care of the healthy voice, and in pathological cases also the physician has often to take a secondary place to the teacher of singing or elocution. There is an old feud between physician and "voice producer," with the result that the natural development of the voice, and the instruction in singing, elocution, and rhetoric, suffer a great deal. We admit that a physician volunteering advice to his patients with regard to "voice production" should not only know thoroughly all the physiological and hygienic aspects, but should also be endowed with the required musical gifts and be well versed in the rules and technic of elocution and singing. If he cannot fulfil these requirements, he should limit himself to advising his patients simply concerning hygienic matters.

And here we are not concerned with the larynx only, but with the entire apparatus of respiration and phonation, as can easily be understood. In examining the body we certainly have to consider the condition of the respiratory organs and that of the resonator, which modifies so largely the timbre of the voice. (See p. 360.) First of all, we must consider whether breathing through the nose is easy and efficient, and then further advice as to mode of life can be given. A person who uses his voice on the stage, as a singer or actor, in public, on the bench, in parliament, in the field, or otherwise, must be informed that unsuitable clothes hinder respiration, and so indirectly weaken the necessary blast of air (tight and high collars, corsets, etc.). Gymnastics, however, reasonably performed, are very advantageous. *Flatau* specially recommends rowing, which is a simple means of allowing a gradual and harmonious increase of energy in all the muscles, and causes the lungs to be well ventilated. Cycling, however, I do not allow under any circumstances, and I disagree herein with *Flatau*, because cycling is always overindulged in and, as *Imhofer* rightly says, the dust-clouds of the streets, however salubrious they may be, are not very beneficial to the respiratory organs. The benefit of hardening, the consequence of excess in alcohol and tobacco, is again worth mentioning. Apropos of alcohol, I should like to say that total abstention from it, in persons who value particularly a glass of wine or beer, is evil advice, provided that it be not contraindicated on account of any chronic catarrh of the upper air-passages. I have never seen any disadvantage from moderate drinking.

The laws of correct respiration and intonation, the exact coördination of the vocal organs, the training and preservation of the voice, the proper use of the different registers—these and other technical questions should be read up in special technical works on the subjects.

SPECIAL SECTION.

I. MALFORMATIONS AND DEFORMITIES.

Malformations and deformities of the larynx are either congenital or acquired. The former are due to anomalies of development, the latter are caused in extra-uterine or later life, as the result of pathological processes in the larynx and trachea or in neighbouring organs, usually resulting in narrowing (stenosis) of both the parts concerned.

1. ANOMALIES OF DEVELOPMENT.

The female larynx is smaller in all its dimensions than that of the male. Nevertheless, men are sometimes found whose testicles have not been fully developed, with a small infantile larynx and consequently with high, boyish voices ("castrate" or "eunuchian" voice); on the other hand, there are not a few women who are possessed of an unusually large larynx and low, deep, manly voices. Defects or the formation of fissures not infrequently occur in the epiglottis, the margin of which then appears notched or bifid. I remember a patient whose epiglottis had the shape of a myrtle leaf. The omega (Ω, jew's-harp-) shaped epiglottis has been already mentioned as a hindrance to laryngoscopy. Many other alterations in shape are not congenital, but are acquired as the result of ulceration or operation.

The thyroid cartilage is sometimes deviated or shows unequal development of its wings, so that the larynx has quite an asymmetrical form. In such a case the rima glottidis is more or less oblique, and this is best seen during phonation. This "physiological obliquity" must not be confounded with a pathological one, seen sometimes in unilateral paralysis of the recurrent nerve. In the latter case obliquity of the rima is caused by the healthy vocal cord encroaching on the middle line during phonation, in order to effect a better closure of the glottis, and this may go so far that an *actual crossing of the two vocal cords takes* place. Such an overcrossing of the vocal cords may also occur, however, under normal conditions, as I have noticed on several occasions.

The arytenoid cartilages then stand, not side by side, but behind one another.

Diaphragma Laryngis.—Sometimes, and nearly always at the anterior commissure *between* the vocal cords, and still more frequently *underneath* them, a membranous fold may be noticed with its free concave edge directed backwards. According to *v. Hansemann*, this congenital formation (laryngeal diaphragm) is the product of intra-uterine inflammation. Other authors maintain that it represents the organised remainder of the epithelial mass, which, according to *Roth*, fills the foetal larynx up to the region where, later, the rima glottidis appears. *Rosenberg* reports of a case of double diaphragm that one of them was expanded between the aryepiglottic folds, and the other beneath it, between the vocal cords. The laryngeal diaphragm does not cause any trouble, especially if the membrane lies below the vocal cords and sinks downwards like a pouch during phonation. If the diaphragm is very large, extending far backwards, phonation and respiration will suffer. The troubles caused by acquired membranous adhesions (through syphilis, diphtheria, scleroma, etc.) are usually much more pronounced, for the membranes are more rigid than the congenital ones and impede, not only the abduction necessary for respiration, but also the adduction of phonation. One case of a congenital membrane on the posterior laryngeal wall has been recorded.

Ventricular Laryngocele.—*Morgagni's* ventricle has been found enlarged like a pouch on one or both sides, and bulging like a hernia into or outside the larynx on pressure or coughing. A supernumerary ventricle (ventriculus tertius) of the anterior laryngeal wall is also on record.

Tracheocele.—Formation of a sac is often found in the tracheal wall. Two forms may be distinguished: in the congenital tracheocele (diverticulum tracheae) a part of the tracheal mucous membrane is seen to bulge or insinuate itself between a gap in the cartilages, or it may be a real rudimentary bronchus, situated as such always on the right side, and showing all the constituent parts appertaining to the tracheal wall. There is also an acquired form of the tracheocele, which is due to injury (punctured wound) of the tracheae; or it is a cystically degenerated mucous gland, occurring sometimes in chronic bronchitis, and is single or multiple.

The diagnosis of tracheocele may be made if we find a soft, round tumour bulging out from the trachea during coughing or on pressure exercised on the trachea; or should such a tumour be already present, it will increase on coughing, and if compressed, will collapse with a sibilant sound.

Treatment.—Many of the malformations described are only accidentally discovered, perhaps at a postmortem. This shows that they need not necessarily be treated. Tracheocele and diaphragmatic formations, if they cause symptoms of stenosis or impede the voice, may be operated upon. It is often sufficient to divide the membrane and afterwards to methodically dilate the larynx. (See later, p. 427.) *M. Schmidt* recommends the removal of the edges of the membrane after division, by means of a double curette, and then to continue with dilatation If the membrane (diaphragm) is not too large, simple dilatation is quite sufficient.

2. STENOSES.

We have already pointed out that congenital malformations (diaphragma laryngis) sometimes also give rise to stenosis, but in the great majority of cases stenosis is acquired as the result of pathological processes in the larynx or trachea in extra-uterine life.

Laryngeal stenoses are almost always due to intralaryngeal causes; extralaryngeal processes very exceptionally lead to stenosis, but tend rather to displace the whole larynx. The trachea, on the other hand, being much more elastic than the larynx, and this elasticity is still more increased by its muscular posterior wall, yields sooner to the pressure of extratracheal tumours, particularly if the latter surround the trachea, which then becomes narrowed. But simple displacement also of the trachea is often observed.

In the following table we have endeavoured to arrange the etiological points into groups in order to elucidate the survey:

(*a*) *Aspiration of Foreign Bodies.*—1. Derived from the outside, *e. g.*, coins, buttons, stones, pins, needles, bones, fish-bones, fruit-stones, teeth, etc.

2. Derived from inside, *e. g.*, blood, food (vomiting during general anaesthesia), pedicled polypi in the neighbourhood of the aditus laryngis, etc.

(*b*) *Injuries.*—1. External, *e. g.*, contusion, fracture, wounds, etc.

2. Internal, *e. g.*, caustics, scalding, operative lesions, etc. Injuries do not often directly cause stenosis, but lead indirectly to it by their consequences, viz., haemorrhage, abscess, oedema, emphysema, formation of membranes, cicatricial contraction, adhesions, etc.

(*c*) *Diseases of the Laryngeal Walls.*—1. Inflammation of the mucous membrane, particularly the phlegmonous, exudative, fibrinous, and subglottic forms of inflammation.

2. Oedema.

3. Perichondritis.

4. Infiltrative and ulcerative processes, *e. g.*, tuberculosis, syphilis, typhoid, malleus, leprosy, scleroma.

5. New-growth.

(*d*) *Granulation and scars.*

(*e*) *Nervous Disorders.*—1. Spasm, *e. g.*, laryngismus stridulus, spasm of the glottis, tabetic crises.

2. Paralyses (bilateral posticus paralysis).

(*f*) *Pathological processes in neighbouring organs* which cause stenosis by pressure on trachea or larynx.

α. Of the aditus laryngis, *e. g.*, inflammation or tumour of the base of the tongue, foreign bodies pressing on the epiglottis, retropharyngeal abscess, and tumour of the laryngeal part of the pharynx.

β. Of the larynx (very seldom).

γ. Of the trachea.

1. Strumous tumour (goitre).
2. Aneurisms (of the aortic arch).
3. Abscess (retrovisceral abscesses).
4. New-growth (of the oesophagus, sternum, vertebral column).
5. Swelling of lymphatic glands.
6. Haematoma or haemorrhage.
7. Persistent thymus.

Pathology.—How stenosis is produced by the above various processes, and in what form it presents itself, will be shown in the following chapters on these special diseases.

Concerning the narrowing or obstruction of the aditus laryngis we refer to Parts II and III of this book. We shall here only deal with those forms of laryngeal and tracheal stenoses which present distinct features as the consequence of previous processes, viz.:

1. Stenosis from granulation.
2. Stenosis from scars.
3. Stenosis from compression.

These three groups really belong to the great department of internal medicine and general surgery, but we will discuss them here because they may come before the specialist for laryngoscopic or tracheoscopic examination.

1. *Stenosis Resulting from Granulations.*—After tracheotomy

we frequently see granulations arising in the subglottic region or in the angle of the wounds, which owe their origin to the irritation caused by a too long or a badly fitting canula. These granulations are apt to hinder the withdrawal of the canula (decanulement), or they may obstruct the trachea to such a degree after the removal of the canula that severe dyspnoea and even death from suffocation might occur. These granulations are prone to be aspirated into the trachea by the current of inspired air, where they are then liable to suddenly swell and become oedematous, as a consequence of the negative pressure present in the air-tubes during inspiration. In other cases granulations are formed, in necrosis of the mucous membrane, as the result of the constant pressure exercised by the canula, or they are the consequence of ulcerative processes, usually of diphtheria. Finally, the granulations undergo atrophy and shrink, leading to stricture and stenosis from the formation of scar tissue (see below).

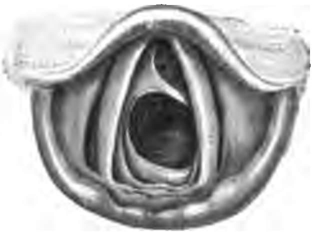

Fig. 133.—Fibrous bands in the trachea (after *Türck*).

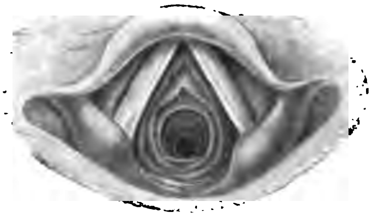

Fig. 134.—Annular stenosis of the trachea (after *Türck*).

We desire to again emphasise that the tracheal cartilages, divided in tracheotomy, are drawn inwards like a valve with the inspiratory air-current, and thus are liable to cause stenosis, more especially so if the incision has been made too lengthy or if the cartilages have become atrophied and softened from prolonged pressure of the canula (chondromalacia).

2. *Stenosis from Scar Tissue* (*Strictures, Fibrous Stenosis*).—In the larynx as well as the trachea strictures occur almost always as the result of syphilitic ulceration. The larynx, however, is much oftener subject to stricture than the trachea. (See Figs. 133 and 134.)

The scars may be seated in the most varied places; they may form a kind of diaphragm between the vocal cords, and further down, deep in the trachea, which they may narrow in an annular manner or obstruct so as to leave only a small aperture. In some patients of my own, who had been the subjects of tracheotomy while still young, the fibrous band projected more or less from the anterior tracheal wall into the lumen. In one of these cases there was, in addition, such a thickening of the epithelium of the posterior laryngeal wall that the lumen was constricted to a small conical opening. Sometimes both organs (larynx and trachea) are affected, and the distortion produced by the fibrous scars might be so pronounced that orientation is rendered difficult.

If the trachea alone is diseased, stenosis mostly occurs above the bifurcation. Tuberculosis, ulceration due to enteric fever or diphtheria, less often give rise to cicatricial contractions and strictures. In the case of enterica it is the perichondritis which leads to stenosis from the formation of extensive scars. Injuries also which have caused considerable lesions are liable to constrict the trachea or larynx by reason of the shrinkage of resulting scars.

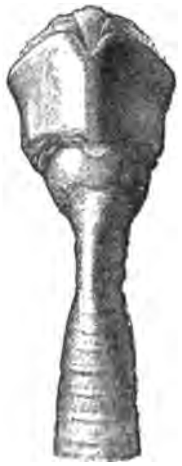

Fig. 135.—Scabbard-shaped compression of trachea (*Hochenegg*).

3. *Stenosis Through Compression of the Trachea.*—In the first line stand here the diseases of the thyroid gland, particularly the cystic goitres, which in regard to their seat give rise to compression of the upper or lower section of the trachea. If both lobes of the gland are enlarged, the trachea is compressed on both sides like a sword scabbard (scabbard form). (See Fig. 135 *et seq.*) The lumen of the trachea then forms a small elliptical space or chink. If only one lobe is hypertrophied, then the corresponding side of the trachea alone is compressed, and, moreover, is also bent angularly—due to adhesions to the neighbouring tissues. (See Fig. 137.) In unilateral struma (goitre) the trachea not infrequently is merely displaced laterally,

without showing signs of compression. The continuous pressure exercised by the tumour finally causes the cartilages to atrophy and soften, and so tends to aggravate the existing stenosis.

Malignant tumours of the thyroid gland are prone to grow into the trachea, as does also carcinoma of the gullet. *Aneurisms* are apt to compress the windpipe, especially aneurisms of the aortic arch on the posterior wall of the aorta, at the spot where the aorta, so to speak, "bridges" over the trachea and left bronchus, and the pulsation of the aneurism is transmitted to the mucous membrane, which bulges and is reddened at the site of compression.

There is no denying that hypertrophy of a persistent thymus gland is liable to produce stenosis. How the sudden death sometimes occurring in small children may be explained by such a compression due to hypertrophy of the thymus we do not pretend to discuss.

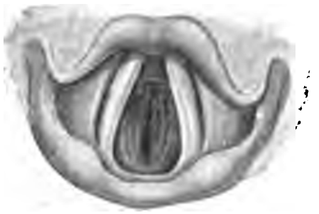

Fig. 136.—Bilateral compression of trachea (image) (*Türck*).

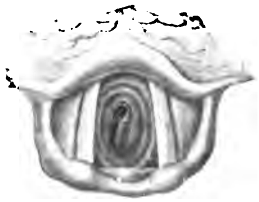

Fig. 137.—Compression of the left side seen as image (*Schrötter*).

Symptoms.—As has been already pointed out in the General Part (see p. 386), stenosis of the windpipe may occur suddenly or in a relatively short time, or gradually. At the same place (see p. 386) the subjective and objective symptoms have been discussed. Here we shall only speak of those symptoms which are not directly concerned with the stenosis, but are in connection with the cause of it, more especially with the cause of compression stenosis of the trachea.

The long-drawn-out course of the recurrent nerves makes it feasible that not only the trachea, but also the nerves themselves, are easily exposed to pressure from diseases in the neighbourhood of the trachea, *e. g.*, in the mediastinum. In the neck both recurrent nerves run in the groove between the oesophagus and the trachea, and are, therefore, better protected against pressure. Hence we may observe that the signs of paralysis of the inferior laryngeal nerves are oftener seen in compression of the intrathoracic part of the trachea, *e. g.*, paralysis of the left vocal cord in aneurysm of the aortic arch. Stenosis *per se* does not alter the voice if it be not combined with paralysis or pathological changes of the vocal cords. In deep-seated stenosis an area of dullness on percussion may be often noticed over the manubrium sterni, corresponding to the primary seat of the lesion and expanding over the vicinity. If the stenosis lasts for a longer time, other signs of disturbed respiration are manifest, *e. g.*, emphysema and atelectasis of certain parts of the lung, hypertrophy and dilatation of the heart, etc.

Diagnosis.—In making a diagnosis we must answer three questions: 1. Is there really stenosis of the air-passages? or, in other words, is the existent dyspnoea to be referred to stenosis? 2. Where is the seat of the stenosis? 3. What is the cause?

The first will be answered without difficulty if the previous history and manifest symptoms are carefully considered (stridor). As regards the second question, as to whether laryngeal or tracheal stenosis be present, this will be safely decided by laryngoscopy or tracheoscopy (indirect and direct method). In many cases external inspection will have already given us the right clue, *e. g.*, if we find a tumour pressing on the trachea. The other external signs, *e. g.*, movements of the larynx on inspiration and expiration and the position of the head are not reliable, because they vary in different cases. I remember two cases of undoubted tracheal stenosis due to goitre which showed very conspicuously the respiratory ascending and descending of the larynx, which is usually taken for a sign of laryngeal stenosis. And, besides, in many cases of short neck, as in women, the movements of the larynx cannot be observed at all; and we should also remember that larynx and trachea may be simultaneously stenosed, as, for instance, as the result of syphilitic processes. The conditions of voice are not always characteristic. Here again laryngoscopy will facilitate the recognition as to whether or not the *stenosis is in the region of the larynx,* and if the larynx is found free, the other methods of examination (*Killian's* direct and indirect method of tracheoscopy) will show us the site of stenosis in the trachea.

V. Eicken maintains that only tracheoscopy alone will admit of exact recognition of seat, size, and kind of stricture (stenosis). Unfortunately, autoscopy requires great technical skill, apart from the fact that it is contraindicated in aneurism owing to the great risk of injuring the aneurismal sac. Severe dyspnoea is very likely to prevent even the introduction of a simple mirror. In such cases one must content one's self with the remaining signs and symptoms, which do not prove to a certainty, but *faute de mieux,* in their entirety, will prove enough for a diagnosis. Lately, the *Röntgen* rays have been resorted to by *Killian,* in order to ascertain tracheal stenosis.

The differential diagnosis between tracheal and bronchial stenosis is not difficult for those who can manage tracheoscopy and bronchoscopy; otherwise an unaided physical examination alone must serve to decide. The "stridor" is less loud in bronchial stenosis than in tracheal stenosis, and both an inspiratory and an expiratory stridor should be *felt* and *heard* at the site of the stenosis. In cases of long-standing stenosis of the bronchus the movements of the corresponding half of the thorax is less marked, and the respiratory sound (bruit) is reduced, the fremitus weaker, and the other lung shows signs of compensatory enlargement (emphysema). (For stenosis due to foreign bodies see Chapter VII.)

Diagnosis.—We must now consider the diagnosis of the primary disease. In laryngeal stenosis laryngoscopy will at once clear up the matter, but, on the other hand, the whole armamentarium of internal medicine will be required to ascertain the nature of a tracheal stenosis. Fibrous strictures are almost always due to syphilis, and *A. Fränkel* contends that syphilis should always be our first thought if we have to deal with a patient who shows the signs of a gradually developed stricture of the trachea. But beware of *mistaking fibrous strictures for stenosis caused by outside pressure.* In the former a *dark hole* *or chink may be seen with white edges;* in the latter, *a more rotund prominence.* (See Fig. 138.)

If we have ascertained that we have before us a compression stenosis, our mind is at once directed to the thyroid gland if the stenosis is seated somewhat high up in the trachea. Deeper seated stenosis points to retrosternal goitre or aortic aneurism. The retrosternal goitre sometimes presents itself in a form that is spoken of in German as "Tauchkropf" (diving goitre), for the reason that the retrosternal thyroid lobe is drawn into the thorax by each inspiration, and ascends during each expiration, and still more so is this the case on coughing, when it becomes visible in the jugular fossa. Naturally, stenosis and dyspnoea are augmented if the goitre slips behind the sternum, for the resistance of the bone only permits the goitre to move backwards towards the trachea, and so the tracheal lumen is usually narrowed until it forms only a transverse chink. Aneurism of the aortic arch often produces paralysis of the left vocal lip; besides, a visible pulsation and an area of percussion dullness over the sternum, an inequality of the radial pulses, and the protrusion of the red and pulsating tracheal mucous membrane seen in the mirror complete the picture of symptoms. *Chiari,* however, reminds us also that, normally, pulsation may be visible on the lower section of the left tracheal wall, and of the spur (see p. 358), but then the redness and protrusion of the mucous membrane are not present, as in stenosis due to aneurism. Malignant tumours grow into the trachea or oesophagus, causing either tracheal or oesophageal stricture, or both simultaneously. On tracheoscopic examination we then find the prominence or protrusion caused by the tumour, the surface of which might be smooth, but is more often uneven, tuberous, or lumpy, and the age of the patient also, and the comparatively quick development of the stenosis will aid us in diagnosis. Benign tumours never perforate the trachea.

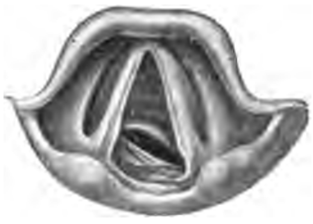

Fig. 138.—Tracheal compression by a retrosternal thyroid goitre. The lumen of the trachea is narrowed to an almost transversal chink by the pressure exercised from in front (*Türck*).

Prognosis.—The danger of suffocation is so much the greater the narrower the windpipe is under normal conditions, viz., in children, and the quicker the stenosis has been developed. But one should not forget that many a gradually increasing stenosis may unexpectedly become urgently acute. For this reason it will be well to be prepared for such a possibility; for example, if we have to treat a patient suffering from tubercular infiltrations or a compressing tumor (goitre).

The dreaded death as the outcome of goitre, which may occur like a thunderbolt from the blue, is not yet quite explained. The patient collapses suddenly on making a rash movement of the head. *Rose's* opinion of this is that the trachea of these patients, owing to the softening and atrophy of the tracheal cartilages, is easily kinked, and that the heart, already weakened by the respiratory and circulatory disturbances, is powerless to overcome the sudden stress.

An intercurrent catarrh may also give rise to an acute attack of dyspnoea. This danger of rapid suffocation is less or not at all to be feared in fibrous strictures. These usually remain stationary, and even in marked tendency to contraction, sudden suffocation hardly ever occurs.

Generally speaking, the prognosis depends on the site of the stenosis and the character of the primary cause. The higher the place of stenosis, the more is it found to be amenable to proper treatment. If dilatation by one or other method fails in uncomplicated laryngeal or high tracheal stenosis, tracheotomy will always save the situation. In acute or rapidly developing stenoses tracheotomy might even establish a permanent cure. In the stenosis gradually produced by some constitutional disease tracheotomy is only palliative. Prognosis becomes worse the farther down the trachea the seat of the stricture may be, and it is bad if the primary cause is of a malignant nature (carcinoma, aneurism, tuberculous glands). Syphilis again offers better chances, as it allows of an antisyphilitic treatment or systematic dilatation.

In any case prognosis quoad vitam et restitutionem is very bad, if in long-standing stricture the lung and the heart have become irreparably affected.

Treatment.—Before all else stands the indicatio vitalis, viz., the prevention of suffocation—prophylaxis. All the other measures as to the treatment of the stenosis itself and its cause come into the second line in point of importance (indicatio causalis).

A. Prophylaxis (Preventive Treatment).—In threatening suffocation, the question arises whether we can expect to remove the stenosing or obstructing factor quickly, by operation through the mouth, so as to reëstablish again the natural conditions and to eliminate the menacing danger. For example, a foreign body so situated that it can be reached with the forceps through the mouth or even with the finger, or if there be an abscess or oedema in the region of the aditus laryngis, is it easily accessible to the knife, or does it take the form of oedema which can be reduced by scarification? in these and similar cases both purposes, the indicatio vitalis et causalis, may be dealt with. In other cases, if the danger of suffocation is imminent, it is our duty to free the air-passage either (1) by operation, involving the opening of the windpipe (tracheotomy), or (2) by other expedient methods, such as the insertion of a tube into the narrowed passage by way of the mouth (intubation or catheterisation).

1. *Tracheotomy.*—Whether we perform superior or inferior tracheotomy, viz., above or below the isthmus of the thyroid gland, depends on the site and character of the stenosis and on the local conditions. If it can be done, the trachea should be opened below the seat of stenosis. Superior tracheotomy, is, however, owing to the lesser vascularity in this area, easier to perform, although the field of operation is limited; yet it should be given the preference, generally speaking, in adults. Nevertheless, great care must be observed not to divide the cricoid cartilage itself, because the "decanulement" later on is long delayed, and necrosis very easily sets in, owing to the pressure of the canula on the rigid cartilage, but which, on the other hand, might result in cicatricial retraction. In children, in whom the thyroid gland is usually higher than in adults, inferior tracheotomy is preferable.

Referring to the text-books on surgery, we desire to emphasise here only certain points.*

The operation should be performed under *general* anaesthesia, because respiration is then quieter, and it also permits a surer operation. If narcosis is difficult to induce, the cutaneous incision, which is the most painful part, may be done under *local* anaesthesia. In cases of urgency, where somnolence has already appeared, one can omit any preliminary anaesthesia, for the sensibility has already become reduced by the somnolence. The incision should be at least 4 cm. (2 inches) long, rather longer than shorter, and strictly confined to the middle line, *while the head must be well retracted.* In superior tracheotomy the cutaneous incision begins from the conic ligament [somewhat below the thyroidal notch (incisura thyroidea)], and must be carried downwards over the isthmus of the thyroid gland. In inferior tracheotomy the incision must begin just above the isthmus, and is continued down nearly to the episternal notch. The superficial fascia must be made tense by means of two forceps, and divided throughout the whole length of the cutaneous incision, or if adequate assistance is not at hand, it must be "snipped" and then divided along a director.

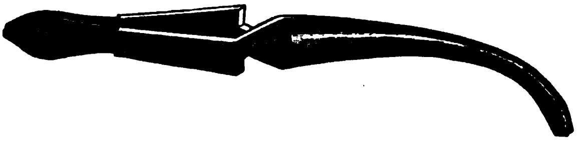

Fig. 139.—Tracheal dilator.

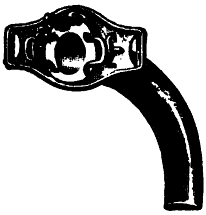

Fig. 140.—Tracheal canula (*Lüer*).

The muscles (sterno-hyoidei, sterno-thyroidei, and the cricothyroidei) are separated, and any bleeding veins carefully ligated. The thyroid gland, which in children is often situated high up, must be carefully separated and pushed downwards, according to *Bose*, by dividing the deep fascia below the cricoid cartilage (fascia thyro-laryngea) transversely by a small incision, and then the gland can be easily separated with a blunt instrument from the trachea, and the isthmus pushed downwards and kept down, in *superior tracheotomy*, and drawn upwards and kept up, in *inferior tracheotomy*. On account of the abundant vascularity and the relation of the anonymous artery, which crosses the trachea, this dissecting and separation must be done very carefully, especially in *inferior tracheotomy*. This done, the trachea appears as a bluish-white band, and all the bleeding vessels having been secured, it can now be opened. The trachea, fixed and lifted up by means of a hook, is punctured at the lower angle of the cutaneous incision and opened up by cutting from below upwards. The edges of the wound are then kept apart by means of a dilator (see Fig. 139) or retractor, and the canula (see p. 418) inserted. The touching of the tracheal mucous membrane always excites some spasmodic fits of coughing, which cause the air to escape with a loud hissing noise, accompanied by some blood-stained mucus. A short pause in the respiration (apnoea) frequently follows the first fit of coughing, especially in patients with considerable stenosis, due, probably, to the suddenly increased supply of oxygen. This pause in the respiration, though short, has a strange, not to say perturbing, effect.

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* The text-books on laryngology, with few exceptions, omit any discussion of tracheotomy and its consequences, whereas they give extensive space to intubation. I consider this quite wrong, and am of the opinion that everybody occupying himself with this specialism and looking up a text-book on laryngology is justified in expecting, at least, that the chief points in an operation which plays so important a part in so many diseases will be discussed, and certainly it is as important as intubation, which is claimed by the laryngologists as their inalienable perquisite.

In cases of urgency and extreme need, tracheotomy must be performed with very primitive instruments, even as *v. Bergmann* hinted, with a pocket-knife. A piece of india-rubber tubing may be used as a canula, threaded at one end, in order to fix it; *hair-pins* may take the place of retractors or hooks. In such a case we must not concern ourselves too much about the bleeding, in order not to lose much valuable time while the patient is in danger of suffocating. Bleeding veins often cease bleeding after the trachea has been opened, and any other vessels may be secured after the operation is finished.

The canula should be large, not too short, and fixed or tied with a tape around the neck; the shield of the canula must be padded underneath with cotton-wool, and the operation finished by packing the cutaneous wound.

If it is at all possible, the trachea should be opened below the stenosis, as, if we do so, the danger of suffocation is at once averted and irritation of the narrowed region by the canula avoided. Unfortunately, in tracheal stenosis situated far down one will often be forced to open the trachea above the stenosis.*

If the tracheotomy is undertaken in order to remove a foreign body or obstructing membranes or any other intratracheal hindrance, simple tracheotomy and a simple canula will be sufficient. In other cases, for example, in substernal goitre, a long canula (*König's* tracheal canula) must be applied (see Fig. 141), or even a catheter, etc.

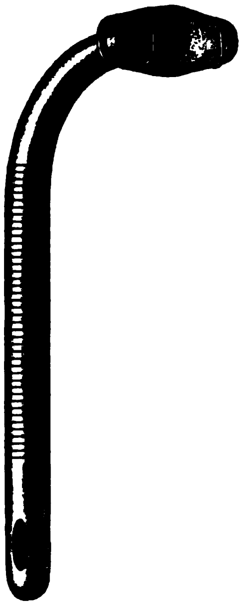

Fig. 141.—*König's* long tracheal canula.

Tracheotomy must be followed by systematic dilatation of the stenosis if the case is a suitable one.

Complications.—Bleeding during and after the operation may become dangerous. Emphysema of the subcutaneous tissue, to a greater or lesser extent, sometimes arises from severe coughing, after blocking, or if the canula falls out, and is of no consequence. Difficulties met with when inserting the canula may be overcome by dilating first the wound or separating the edges by means of dilators or retractors. More unpleasant are the complications caused by the pressure of the canula or as the result of delayed "decanulement." Generally speaking, the canula should remain for at least two days, but not longer than five days, continuously; the inner canula, however, must be changed daily, so that no obstruction may occur. If the canula remains in the wound for too long a time, if it fits badly, or it has a sharp edge somewhere, it will lead, by the pressure it exercises, especially about the stenosed part, to necrosis of the mucous membrane, *i. e.*, *decubitus*, which manifests itself by pain, fever, and blood-stained expectoration. In such a case matters may be sometimes amended by changing the canula for a shorter one, or by replacing it by a soft tube, or even a drainage-tube. A badly fitting or irritating canula may also give rise to granulations, and so again to stenosis, which may render the removal difficult. (See p. 409.) The removal of the canula ("decanulement") may also prove difficult on account of deviations of the trachea or displacement of some cartilages or parts of cartilages, due to faulty method of operation. These faults must be removed by surgical means. Granulations may be cauterised by nitrate of silver, the curette, or snare.

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* Where the cause of stenosis can be removed by a radical operation (as in goitre), this operation should be taken into consideration.

Final "Decanulement."—The complications mentioned above, and which cannot sometimes be avoided in spite of the greatest care, make it desirable to finally withdraw the canula as soon as possible. This is especially so in a case where the stenosis has again become dilated, so that the air-current is now rendered sufficient. That the stenosis has become more free can be proved by closing the canula temporarily with the finger or with a cork, while the patient is required to breathe quietly. In nervous patients and in children it not infrequently happens that a fresh attack of dyspnoea at once occurs on probatory closure, not perhaps because of the stenosis still persisting, but because of the psychic reflex irritability. *M. Schmidt* explains this recurrent dyspnoea as being due to spasms. Children have frequently lost the habit of breathing through the glottis and of automatically abducting the vocal lips. Here, an intubation once performed will succeed in reinducing normal free respiration. Premature removal of the canula is a mistake, and must be avoided in order not to again produce the preexisting dangerous conditions. The canula being finally withdrawn, the cutaneous wound soon closes under a protecting bandage, and fistulas which may persist may be caused to close by refreshing their edges and sewing them together.

2. *Intubation and "Catheterisation" of the Air-passages.**—

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* It would be well to drop the term "tubage" in preference for "catheterisation" in order to avoid confusion of the terms "tubage" and "intubation."

Intubation, after the method of *O'Dwyer*, is performed under the guidance of the finger with straight metal or vulcanite tubes; while *catheterism*, after *Schrötter*, is executed with the aid of the mirror by means of catheter-like tubes of vulcanite. *Intubation* is chiefly applied in stenosis from acute inflammations, as in diphtheria, hence it is mostly used in children. *Catheterisation*, on the other hand, is chiefly concerned with adults. But we desire at once to say that *O'Dwyer's* tubes, in suitable sizes, are also occasionally used in adults for acute inflammatory stenoses, and that both kinds of tubes might be introduced into the larynx for the purpose of dilating chronic stenosis. One should be very careful in introducing tubes into the windpipe in cases of acute inflammation, as the pressure caused by the tube upon the most irritable mucous membrane is apt to aggravate the inflammation, or even to cause the inflammatory process, which was perhaps already abating, to become aggravated. An exception therefrom, however, must be made in diphtheria, which, as we have said above, is commonly and chiefly treated by *O'Dwyer's* tubes, but here also we must take into account a possible negative result, and must, therefore, be prepared to perform tracheotomy after any intubation if necessary.

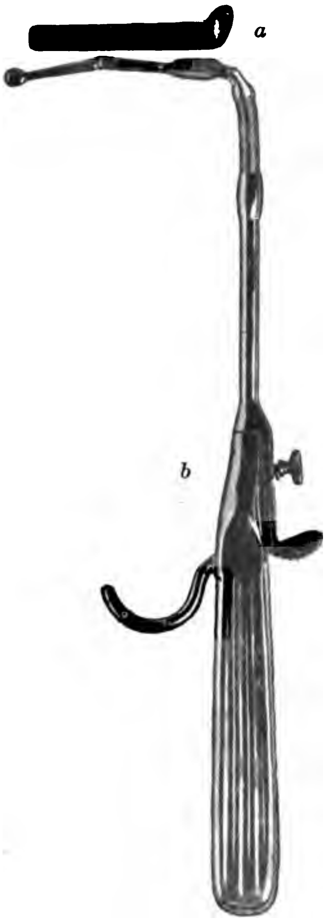

Fig. 142.—Instruments for intubation (after *O'Dwyer*): *a*, Tube; *b*, introducer with the obturator (Ger., *Mandrin*).

(*a*) *Intubation of the Larynx After O'Dwyer.—In children*, it is performed with slender oval tubes, the size of which is adapted to the age of the child. *In adults* tubes of vulcanite are chosen because metal tubes would be felt to be too heavy. The one end of each tube is broadened like a *skull,* and it is provided with an obturator (Ger., *Mandrin*), which projects from the other (lower) end so as not to injure the mucous membrane when the tube is introduced. The intubation is done in the following way: The child—we will suppose that our patient is a child—is held firmly (as is described on p. 112), and its mouth kept open, if need be, by a gag.

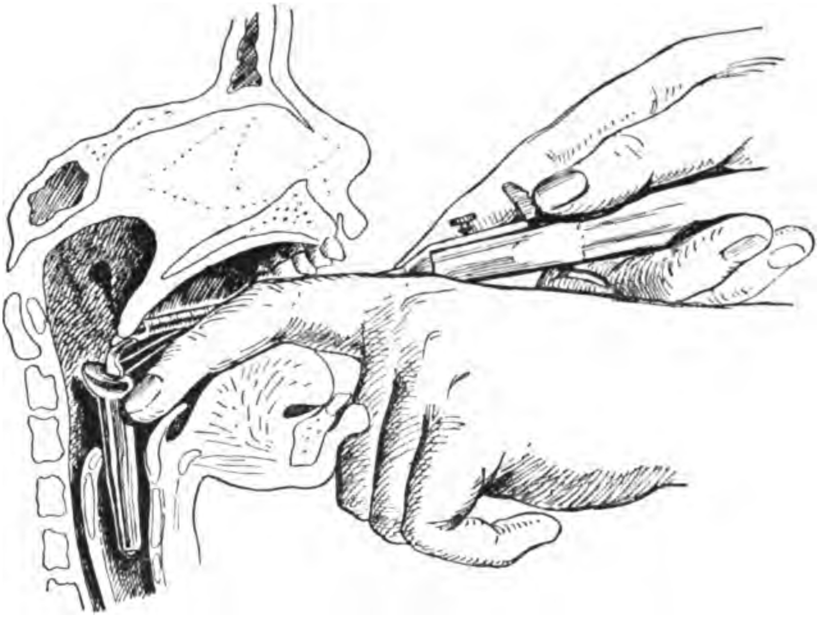

Fig. 143.—Intubation (after *O'Dwyer*) (schematic).

The left forefinger is inserted behind the epiglottis and pressed forwards against the base of the tongue (see Fig. 143), then a tube of suitable size, the head of which is threaded with a double silk thread, is introduced by means of an introducer, always following the left forefinger likewise behind the epiglottis (see Fig. 143); the handle of the introducer is now slightly elevated, so that the end of the tube does not slip backwards and downwards into the gullet; and the tube is then gently inserted forwards and downwards into the larynx between the rima glottidis. While the left forefinger fixes the head of the tube, in order to prevent its sliding backwards, the introducer, together with the obturator, is now withdrawn through the mouth. In order to free the introducer together with the *mandrin*, it is necessary to push the bolt fixed to the handle of the introducer forwards. If the tube is in the right position, one may at once recognise it by the hissing noise caused by the respired air, and also by the respiration itself becoming quieter. If this is the case, the skull-shaped broadening of the head of the tube then lies above the glottis and on the ventricular folds; whereas the smaller neck of the tube below the head lies between the vocal cords, and the broader body of the tube lies below them. If the dyspnoea does not decrease, then the tube has been seemingly inserted into the oesophagus,* and in such a case the tube must be withdrawn by the silk thread and then reintroduced. The tube being now in the right position, the silk thread is tied to the ear, or, by means of adhesive plaster, to the cheek. In order to remove the tube, if the little patient has bitten through the thread, an extractor must be used. This is an instrument shaped like laryngeal forceps, which is inserted in the same manner as the introducer, with the blades closed, behind the epiglottis, and then into the head of the tube. A little pressure on the lever of the instrument is now sufficient to open the claws, and so to grasp the tube and extract it. (See Fig. 144.)

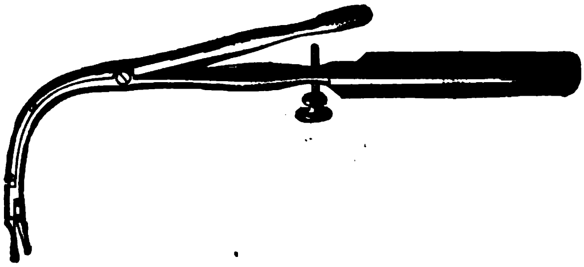

Fig. 144.—Extractor for *O'Dwyer's* tube.

The entire manipulation requires some technical skill, and it is particularly difficult to pilot the claws of the extractor into the tube. The extractor, however, is indispensable in cases where the threads, as has been said, are bitten through or have had to be removed by the physician himself, on account of the child continually tugging at them. This may also be prevented, as is advised by *Ganghofer*, by tying the hands of the child.

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*Translating editor's foot-note: Or, as sometimes happens, the tube has been inserted between the tracheal wall and the diphtheritic mucous membrane.—F. W. F. R.

The tube should be watched by the physician or by trained nurses, and left in the larynx as long as respiration remains free; but under no circumstances should it be left there longer than twenty-four to thirty-six hours, because necrosis and ulceration are easily produced by the pressure of the tube on the vocal cords, which in turn may lead to extensive destruction, resulting finally in stenosis. If, after withdrawing the tube, dyspnoea recurs, or if it has already shown itself before extubation, then intubation must be repeated. If several reintubations are made without the wished-for result, then tracheotomy must be performed.

Complications.—Owing to unskilfulness on the part of the operator, injuries of soft parts are not infrequent; but even the most skilful and careful manipulations, however, may not prevent diphtheritic membranes being separated and pushed on ahead by the advancing tube, and these membranes are then liable to block the trachea to such a degree that tracheotomy must be quickly performed. Having inserted the tube without any injury and accident, coughing, however, may sometimes be so violently excited that a tube which does not exactly fit may be flung out. At the time of the attempt to withdraw the tube, it may often happen that it is pushed into the trachea, and this in particular happens if the tube was smaller than the lumen. If this accident occurs, tracheotomy must be performed without delay. Erosions and ulcers caused by the pressure of the tube have been mentioned. Very unpleasant sometimes is the difficulty of feeding children while the tube is inside the larynx. The children often "swallow the wrong way," and the fluid, which easily reaches the deeper air-passages by way of the tube, may excite violent paroxysms of coughing, and even pneumonia has been observed to follow the misswallowed food.

Although some of these evils may be avoided by skill, care, and experience, yet there are many disadvantages—not the least of these is that the installed tube has to be constantly and permanently watched—which would justify the raising of the question as to whether tracheotomy in the long run would not be preferable. Surgeons will certainly answer in the affirmative, though tracheotomy itself is not at all free also from unpleasant incidents. On the other hand, it should be remembered that intubation is a bloodless operation, for which permission is readily given; also that it requires much less time for recovery, and that its results are better in the stenoses due to diphtheria —at least, according to American and German statistics. But again we desire to remind that usually only in the severe cases of diphtheria, where the process is already far advanced and has spread into the deeper air-passages, is tracheotomy strongly indicated. Generally speaking, in threatening stenosis of the lower air-passages tracheotomy is preferable, and for the reason that the stenosed region is rendered directly accessible by opening the trachea, and the opening itself, moreover, may be easily dilated if this is required.

Intubation is contraindicated—(1) In the case of foreign bodies, for these would only be driven further down into the trachea; (2) in oedema of the larynx, because the swollen parts are likely to cover, and thus obstruct, the head of the tube; (3) in tuberculosis, because in the beginning (stage of infiltration) the conditions are mostly aggravated, and later, if fibrous stricture has developed, we may have to reckon with the exacerbation of old encapsulated abscesses.

(*b*) *Catheterisation of the windpipe*, after *Schrötter*, is performed with suitably curved tubes of vulcanite. (See Fig. 145.) These tubes have a length of 26 cm., and their oral end is circular, while their laryngeal end corresponds to the shape of the glottis and is triangular, and shows, like all catheters, two oval eyes. Having previously anaesthetised the larynx, the catheter, under guidance of the mirror, is introduced into the larynx in the ordinary manner (p. 383). It will be necessary, however, to press slightly in order to get past the stenosed site, and here also one may recognise that the catheter is in the right position by the hissing noise of the escaping air; and that it has not slipped into the gullet by mistake. At the beginning of the treatment the tube only remains for some minutes, later on, for an hour or longer. The sensitiveness of the mucous membrane soon yields, so that later anaesthesia becomes superfluous. Many patients, indeed, learn to catheterise themselves.

Complications are mainly the same as in intubation, but as the catheter only remains for a short time, they are far less serious. Here also, again, tracheotomy will sometimes be necessary.

B. Causative Treatment.—If there is no immediate danger to life, one should always endeavour to attack the stenosis or its cause directly. This may occasionally be done by drugs (caustics, for example), but usually much better by operation.

(1) *Treatment by Drugs.*—The greatest contingency for this treatment is furnished by the syphilitic stenoses. Even in very long-standing narrowing of the larynx or trachea caused by syphilitic ulceration a trial with antisyphilitic treatment is not only justified, but also useful, because a gummatous infiltration may be present, which exercises a certain influence upon the stenosis. In stenoses due to goitre of slight degree, iodine, given internally and externally, may prove beneficial. The preparations of the thyroid gland, praised so enthusiastically, are less efficient than the iodides and iodo-vasogen (6 to 10 per cent.), the use of which I have tested, both internally and externally (see p. 327). If the troubles increase, however, nothing but an operation will be of any avail. In aneurisms, which sometimes also originate from a syphilitic degeneration of the vessels, the iodine and mercurial treatment may likewise be undertaken. *J. Fränkel* reports several successes he achieved by this treatment, either by relieving the symptoms or even

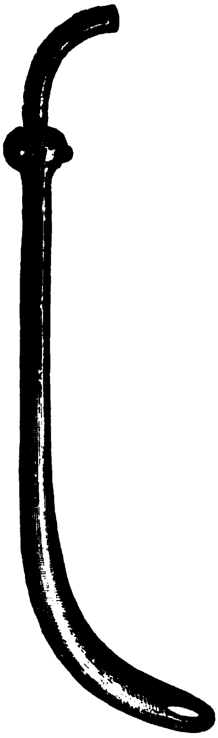

Fig. 145.—Tube for catheterisation (after *Schrötter*).

removing them altogether for a time. In some cases of aneurism iodine may be continuously given (mornings and evenings, 0.25–0.5 sodium iodide for three weeks and repeated) and a suitable diet (milk, vegetables, white meat) may be able to arrest the enlargement of the aneurism.

In desperate cases morphine must be administered.

(2) *Surgical Treatment.*—Here extra- and intralaryngeal operations are performed. The extralaryngeal and extratracheal operations leave the larynx and trachea intact, as is the case in operations for goitre; or one or the other of the two organs have to be opened, as in laryngotomy or a tracheoplastic operation.

Intralaryngeal and intratracheal operations vary very much according to the site and kind of the disease. If one needs to operate for intratracheal hindrances, such as fibrous bands, scars, or polypi, etc., *direct superior* and *inferior tracheoscopy* will prove themselves very useful, according to the method employed, viz., if one desires to operate through the mouth or from the tracheal wound. Further remarks thereon will be found in the special chapters. Here we wish to speak only of the method of dilatation of chronic stenoses, which is different, just accordingly as tracheotomy has been previously performed or not.

(*a*) *If tracheotomy has not been performed on patients, Schrötter's* method of catheterisation, as has been described above, is the rule, although no insurmountable objection against *O'Dwyer's intubation* can be advanced. At the commencement, soft catheters, armed with an obturator, should be used, because their introduction is much easier; later on, more rigid tubes of vulcanite must be used. Gradually, thicker catheters are employed, and they may at first remain in position for a few minutes; later on, for half an hour or longer. Increasing the size (number) of the catheter too quickly may be followed by very untoward incidents, and if such be the case, catheterisation must at once be stopped. Having dilated the lumen to a certain size, catheterisation should yet be continued, at intervals, for a shorter or longer period, so as to prevent any relapse. Such relapses frequently occur in stenoses due to shrinking processes.

It is very convenient, if the patient himself learns to introduce the tubes; this, however, is only possible in *Schrötter's* method, but never in *O'Dwyer's*. The *O'Dwyer's* tubes should always be withdrawn, if possible, by the silk threads attached to their heads, as this is a much more simple procedure than the use of an extractor; besides, in adults, we need not be afraid that the thread will be detached. In stenoses situated far down, *Schrötter's* tubes of vulcanite must be elongated by means of stretching them in hot water, in order to insure that the catheter has reached as far as the narrowed region, when passed from the mouth.

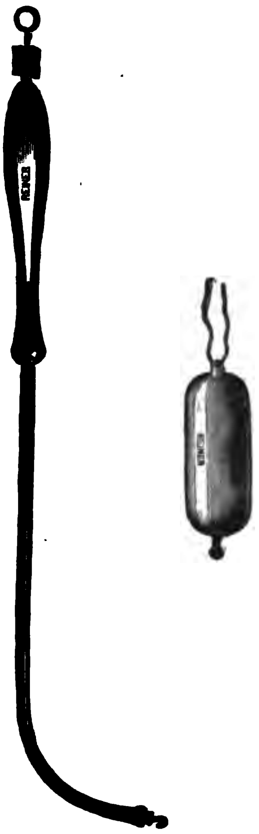

Fig. 146.—Tin dilator (bolt) for obturation of the larynx and introducer (*Schrötter*).

(*b*) *If tracheotomy has been performed,* the stenosis (if laryngeal) may be dilated by way of the mouth or through the wound.

In order to dilate *through the mouth, Schrötter's* dilators of *tin,* which are intended to gradually dilate the larynx (obturation of the larynx), are introduced. The tin dilators (bolts) fitted with an eye on the upper, and with a knob on the lower, end (Figs. 146, 147, 148, 149) are introduced in the same manner as the tubes, under guidance of the mirror or finger, into the larynx by means of a special introducer, until the knob (button) appears in the canula, which is fenestrated in its upper wall for this special purpose. The knob is retained in this position by means of a minute pressure forceps, or, after *Heryng,* by a small slot in the upper wall of the inner canula. This latter (inner canula) is inserted by *Heryng* after the knob of the tin dilator has passed through the eye in the wall of the outer canula. The eye of the dilator is armed with a silk thread, which hangs out from the mouth. The dilator remains for twenty-four hours if the patient is not hindered in swallowing.

Through the tracheal wound, screw-shaped dilators are introduced (very uncomfortable), or chimney or T-shaped canulas. In using the chimney canula, the chimney piece is first introduced, and then the tracheal canula is inserted through it into the trachea; in the T-shaped canula, after *Dupuis*, both halves are each inserted separately, and then joined together in situ.

If the stenosis is below the wound, it may be dilated through the wound with catheters or tubes in the manner described above.

Generally speaking, the effect of this systematic dilatation of very narrow fibrous stenoses is not too promising, apart from the fact that the patience of both the physician and the patient is rather much tried; relapses frequently occur just when the treatment has come to an end. Finally, we may come to the conclusion and advise the patient that he would be far better off if he permitted an operation, so that the stricture could be excised from the outside, and subsequently catheterised, if this be found necessary.

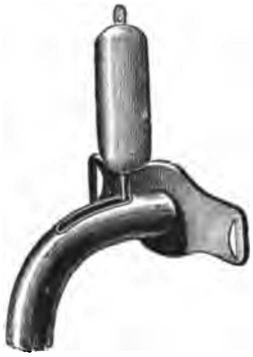

Fig. 147.—Canula fitted with *Schrötter's* tin bolt.

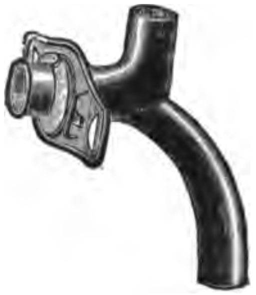

Fig. 148.—Chimney-canula (after *Dupuis*) (one-half natural size).

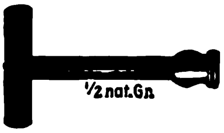

Fig. 149.—T-shaped canula.

II. ACUTE LARYNGITIS.

1. ACUTE CATARRH OF THE LARYNX (LARYNGITIS ACUTA CATARRHALIS).

Etiology.—Acute catarrh of the larynx arises from the same causes as do acute rhinitis and acute pharyngitis. Here, as well as there, chemical, mechanical, and thermal irritations are concerned. "Colds" play an important rôle, at least inasmuch as they prepare the soil for the invasion of the bacterial intruders. I do not propose to here again repeat what has already been said in the discussion of all these factors in the chapters on the various diseases, and only desire to say a few words on some features peculiar to the etiology of laryngeal catarrh, *i. e.*, the overstraining of the voice professionally or on certain occasions.

Laryngitis occurs, as does also rhinitis, in the course of infectious diseases. This uniformity in the etiology serves to explain the fact that catarrh of the larynx is so often seen in association with nasal and pharyngeal catarrh. It is then spoken of as descending catarrh of the upper air-passages, which starts in the nose, and gradually spreads downwards step by step. Strictly speaking, one should call it secondary laryngitis, though it would be difficult to define it clinically, for all the three forms of catarrh may occur simultaneously. On the other hand, it cannot be denied that isolated laryngeal catarrh occurs, *e. g.*, after overstraining the voice. The bulk of the cases, however, partake of the nature of a diffuse catarrh of the upper respiratory tract, the symptoms of which show themselves at the seat of the locus minoris resistentiae; sometimes in one, sometimes in the other, organ or part of an organ. Usually, the catarrh does not stop at the larynx, but continues onward into the trachea and bronchi, and very often the signs of a laryngo-tracheitis are still present when the catarrh of the upper tract has for some time subsided. In some cases, perhaps in subglottic laryngitis, the process may have passed from the trachea into the larynx.

Men are usually more prone to laryngeal catarrh than women, because they are more exposed to professional and trade injuries, and indulge much more in the abuse of alcohol and tobacco.

Pathology.—Inflammation of the laryngeal and tracheal mucous membrane shows different degrees of extent and intensity. The slighter degree is characterised by hyperaemia of the mucous membrane and scanty secretion; the more severe form, by reddening, and frequently, though not always, by swelling of the mucous membrane with hypersecretion and loosening, or actually desquamation of the epithelium. In some cases haemorrhages may even be noticed. We may mention as a fact worth remembering that the infiltration of the tissues with the inflammatory products may go on to acute oedema. It is probable that in such a case not only the mucous membrane, but the submucous tissue, is also affected. In diffuse inflammation of the laryngeal mucous membrane the redness is evenly distributed, though it is more conspicuous in the region of the vocal cords, because otherwise these contrast strongly by their white, glistening colour. Circumscribed inflammation, as can easily be understood, occurs in those parts which are most commonly exposed to noxious influences, *i. e.*, vocal cords (chorditis), and more seldom the epiglottis (epiglottitis). Not infrequently the lower (under) surface of the vocal cords, either alone or in association with other parts of the larynx, become inflamed (subglottic laryngitis). The secretion here, as a rule, is scanty, mucoid, and glassy. In more severe inflammation the secretion is more abundant, turbid, yellowish green, and dries on some places, forming crusts and scabs, which may here and there show blood-stains. In the latter case the laryngitis is spoken of as *dry haemorrhagic laryngitis* (laryngitis haemorrhagica sicca). This arises chiefly, in my experience, on the basis of former pathological changes, such as are found in persons suffering from atrophic rhinopharyngitis, *i. e.*, it is mostly an acute exacerbation of a long-standing chronic catarrh. The secretion adheres so firmly in this form of laryngitis that patients experience great difficulty in bringing it up, and consequently this occasionally causes a little bleeding, which stains the expelled mucus and scabs brown. For the macroscopic changes, we refer the reader to the description of the laryngeal mirror examination (see below). Microscopically, one finds the signs of inflammation, viz., round-celled infiltration, hyperaemia, dilatation of vessels, etc.

Symptoms and Course.—These differ according to the severity of the case.

In the mild cases the onset is usually sudden; there is a sensation of tickling and soreness or irritation in the larynx, which causes the patient to cough or even aggravates actual paroxysms of coughing. The secretion is scanty: only a little glassy and viscid mucus is brought up, or secretion may be entirely absent. The voice sounds impure and hoarse, and soon becomes fatigued. The general health is but little disturbed, and the laryngitis, after a few days, usually subsides.

In the more severe cases all the symptoms described above occur, but are so much increased as to occasion painful sensations. The patients are much troubled by a feeling of "rawness," of burning and smarting in the larynx, and, if the trachea also is affected, the sensations are referred to the upper part of the sternum. There is a short dry cough, especially aggravated at night-time. At the onset here, also, the secretion is scanty, but later on it increases in quantity, and the expectorated mass may be mucoid, mucopurulent, and sometimes even blood-stained. The voice is always disturbed, and all the stages from hoarseness to complete aphonia may be experienced. Patients complain that "something sticks in the larynx," and that speaking and eating are painful. The general health is often disturbed, and the entire course of disease extends for about three weeks or more, and the voice especially is disturbed for a very long period.

It is unnecessary to insist that both forms of laryngitis are not really distinct or separate varieties, but that they merge one into the other; hence the great diversity of the laryngoscopic images.

In the mild cases the mirror shows changes, mostly in the region of the vocal cords only. The vocal cords appear diffusely reddened, or only on the edges or in patches. *In severe laryngitis* the vocal cords are altered into thick, red, fleshy pads, which appear at first dry, and may even seem to be fissured, while later they look more humid and glistening. The ventricular folds also are swollen, sometimes to such a degree that they entirely conceal the vocal cords underneath, or leave only a small edge of them free. The thickening of the ventricular folds is not always equal on both sides, so that the vocal cords on one side may be visible to a greater extent than on the other side. If the process is limited to the epiglottis (epiglottitis), the cartilage loses its graceful shape and becomes clumsy, swollen, and thickened, and greyish or greyish-yellow follicles become visible at the margins.

In cases of severe diffuse laryngitis the mucous membrane is widely reddened and swollen, and even oedematous at some spots; and the posterior laryngeal wall bulges forwards so much that the vocal cords cannot be approximated during phonation. The voice, moreover, may be altered, for the reason that the muscles or groups of muscles become involved by the inflammation. They may become oedematous with the transudation (oedema; anasarca), so that they are unable to properly perform their function.

Incompetency of the internal arytenoid muscle is the most common (paresis of the "internus"); and in this case the vocal cords do not form a straight line, but show a concave or scythe-like margin. The glottis on intonation appears as an *elliptic* chink. Less common is incompetency of the "transversus" and "obliquus"; and in this instance the *posterior part of the glottis* gapes in the form of a *triangular space.** Both kinds of pareses may be present in combination. The vocal cords are here and there covered with small masses of mucus, mucopurulent or glairy secretion, which not infrequently stretches from one vocal lip to the other, forming a sort of mucous bridge.

Laryngitis haemorrhagica sicca and laryngitis subglottica require separate notice.

1. *Laryngitis Haemorrhagica Sicca (Dry Haemorrhagic Laryngitis).*—As regards its pathology, we must refer to what has been said previously. (See p. 432.) The clinical symptoms here are very marked. There is a troublesome sensation of dryness and irritation, causing cough, and the patients not infrequently complain of pain and dyspnoea. The mirror shows

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* See the corresponding diagram in Chapter X, Figs. 206–209.

the vocal cords to be much reddened, as if varnished, and covered at some spots with discoloured brown scabs. Owing to the dryness and friability of the mucous membrane, its epithelium is liable to desquamation, especially at the places where it is more exposed to mechanical irritation, *i. e.*, on the edges of the vocal cords, on the vocal processes, and posterior laryngeal wall. On these places small erosions or ulcers may occur, which have been improperly termed "catarrhal ulcers," so that the vocal cords may become serrated as the result of these ulcers.

Usually, these erosions or ulcers, which, however, are also found in ordinary catarrhal inflammation, soon heal up, or they are overlooked because of their insignificance. It is necessary to use a very intense light (incandescent gas or sunlight) in order to discover them, and then they are only recognised as small dimples, which are veiled as if with a grey or opaque deposit. In some cases they are more distinct and contrast much better with their red surroundings.

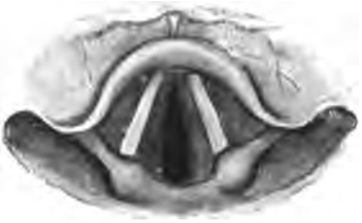

Fig. 150.—Subglottic laryngitis

2. *Laryngitis Acuta Subglottica* (*Subglottic Laryngitis*).—Here the inflammation is limited more to the under (lower) surface of the vocal cords. (See Fig. 150.)

Pads are formed which sometimes project somewhat more into the lumen than the vocal cords themselves. The glottis appears as if divided into two stories, and on lateral examination (subglottic examination), moreover, a groove can be seen which separates the upper from the lower surface of the vocal cords. This subglottic swelling is mostly met with in children, in whom it occasionally gives rise to nocturnal *attacks of stenotic dyspnoea*.

Children who, save for some slight catarrhal irritation, have felt quite well during the day, and have gone to bed calmly and quietly, suddenly wake up with all the signs of dyspnoea, stridor (see p. 386), and violent raucous coughing. The entire alarming scene lasts about half an hour or more, or even two hours, and then the child breaks out into a copious perspiration; the dyspnoea and respiratory oppression subside, and the whole attack is over, sometimes forever, but usually only to be repeated on the following night. There are children, especially those of lymphatic appearance, with hyperplasia of the pharyngeal and palatine tonsils, who are frequently visited by such alarming attacks. This ailment of children, originating in the subglottic swelling described, the symptoms of which are similar to those of real diphtheritic "croup," has been termed "*pseudo-croup*." This much, however, is certain, that not only the *subglottic swelling, but every other kind of swelling* in the region of the larynx, is prone to excite these attacks of "pseudo-croup" in children, on account of the natural narrowness of the infantile larynx, though I do admit that the "croupous cough" is more liable to occur in subglottic catarrh than in other affections of the larynx.

It is not altogether beyond doubt whether the mechanical impediment of respiration, due to subglottic swelling, is sufficient to excite the nocturnal attacks of "pseudo-croup." It may be quite possible that just in the recumbent position—exactly as in the case of the nasal cavernous tissue—some kind of infiltration of the mucous and submucous tissue occurs, which causes transient obstruction of the respiration. The suggestion that the secretions are more liable to dry during the night, and so consequently would block the rima glottidis, is somewhat far fetched. It is rather due to the greater dryness of the mucous membrane at night that a spasm of the glottis is excited by a reflex,—more especially in lymphatic and irritable children than in others,—and that the loud cough, as *Gottstein* points out, may be explained by the explosive expiratory separation of the spasmodically closed glottis.

Laryngitis subglottica is, in my experience, not infrequently met with in adults as part also of a general catarrhal laryngitis. This might in some cases, perhaps, be due to inflammation spreading more deeply, *i. e.*, into the submucous tissue. (See p. 439.)

Other chronic processes also, such as tuberculosis and scleroma, are often associated with subglottic inflammation.

Diagnosis.—Although the anamnesis and symptoms alone will very often permit of diagnosis, only the mirror will show us the exact extent and intensity of the disease. The subjective and objective symptoms, however, need not always be in direct proportion to what we find by the laryngoscope. One person will speak well, though his vocal cords may be very red, and another will complain very much of functional and other disorders yet, we may not be able to discover any very marked changes. *M. Schmidt* contends that the functional disturbances in the latter case are due to muscular incompetency. (See p. 556.) In some cases the diagnosis is less easy if there are erosions or if only certain circumscribed portions are swollen. If there is only a *unilateral swelling of the vocal cords, the disease is usually not a simple catarrh.* Generally, catarrhal laryngitis does not often lead to defects of any very marked depth, and we should bear in mind that such *deeper affections are much more frequently caused by tuberculosis, syphilis,* and similar "dyscrasias," etc. The greatest difficulty that we experience is met with in young children, because of the resistance they offer to laryngoscopy, although we may succeed much oftener than we expect. During an attack of dyspnoea introduction of a mirror is, of course, forbidden. From the attack itself and the differential diagnosis we will be able to decide whether we have to deal with a case of "pseudo-croup" or diphtheria, and the diagnosis, therefore, must be made by other means than by the mirror. If the relations report that the little patient was quite well before the nocturnal attack, if we do not find the ominous false membranes in mouth or throat, and if the lymphatic glands are not swollen, then it is probably a case of "pseudo-croup" with which we have to deal.

Prognosis.—The prognosis of acute laryngitis is usually good, though the tendency to pass into a chronic laryngitis is not excluded, more especially if the patient is careless and the disease frequently recurs. The alarming pseudo-croup also permits a favourable prognosis.

Treatment.—As regards general treatment, the same rules are available as for acute rhinitis. In the more severe cases, however, the patient should remain at home, and if there be fever, should be kept in bed. In mild cases these measures are not necessary, but in inclement weather the patient is certainly better at home. For the rest, sweating or *Priessnitz* cataplasms may be ordered, and the patient advised to speak as little as is necessary, and then only in whispers, and tobacco and alcoholics also must be discontinued. Beverages ought to be neither too hot nor too cold, and in severe inflammation ice at the beginning is very useful. As elsewhere, so in these matters concerning laryngitis, experience teaches. In marked irritation narcotics, and in dry catarrh expectorants, must be prescribed. I prefer in the latter case to prescribe, according to *Eichhorst*, an infusion of ipecacuanha with iodide of potassium, and later on, either morphine or codeine. (See p. 393.) In order to stimulate secretion, inhalations will be found beneficial. (See p. 398.) Some patients again experience marked discomfort from inhalations, and also cough more. If this be so, then inhalations must be discontinued. On the whole, it is well to abstain from ordering and prescribing too much, as in any other acute inflammation, for it is very likely to irritate the more and to only retard recovery. Gargles, which have, so to speak, become traditional in the treatment of acute pharyngo-laryngitis, must be forbidden, because they interfere with the repose of the larynx, apart from the fact that gargles never come into contact with the laryngeal mucous membrane.

Many cases of acute laryngitis recover without any local treatment. If, after the subsidence of the acute stage, there are still some remaining troubles, such as burning or irritation, or should the mucous membrane not assume its former appearance, only then may one try to paint or brush the mucous membrane over with a 2 per cent. or 2½ per cent. solution of silver nitrate twice or thrice *per week;* and should this fail, then resort must be had to stronger solutions. For the crusts and scabs, the application of iodide of potassium is best. A weak solution, as is used for pharyngitis, is preferable. (See p. 253.)

In children acute laryngitis ought to be treated somewhat more actively. Under all circumstances they must be kept in bed, and the room ought to be well ventilated and moistened by means of boiling water or by sheets soaked in water and hung up. *Priessnitz's* cataplasms should be ordered only where it is possible to keep them in the right position, and inhalations should not be ordered at all. For beverages, much warm milk, with or without mineral water, and lemonade, etc., are most suitable.

If it is necessary to prescribe something, an expectorant or a weak solution of iodide of potassium may be given, and in cases of marked irritation, codeine may be ordered.

An attack of "pseudo-croup" is best treated by local derivatives, viz., hot sponges placed on both sides of the neck. Of course, the water used for the sponges must not be so hot as to cause any scalding. If one can induce the child to partake of really warm drinks, then this is also apt to shorten the attack by stimulating the perspiration. Emetics are superfluous, and intubation or tracheotomy should never be resorted to, as the prognosis is always good.

2. PHLEGMONOUS LARYNGITIS.*

Etiology.—Phlegmonous laryngitis, which not only involves the mucous membrane, but chiefly the submucous tissue, is due to infection by pyogenic bacteria which have immigrated from the surface or neighbouring organs, or even from far-distant parts of the body. If we notice distinct gaps in the epithelium of the mucous membrane, we should not hesitate to assume that infection has taken place from the surface. Thus is explained the phlegmonous inflammation which sometimes occurs subsequently to tuberculous, diphtheritic, or typhoid ulceration, or after injuries or burns. We should not, however, forget that even minute lesions of the epithelium, unrecognisable by the laryngoscope, are capable of admitting inflammatory germs which may enter the larynx by air-current or otherwise. The various stimuli which play a rôle in the etiology of simple laryngeal catarrh tend to foster the entrance of micro-organisms. A priori, it may be assumed that every laryngeal catarrh is capable in due course of spreading into the submucous tissue, and this fact is corroborated by experience. One frequently sees, in persons suffering from laryngeal catarrh, who maltreat their voices and smoke and drink excessively, that their catarrh becomes worse, which fact points to a spreading of the process into the deeper tissues. The deeper spreading of the inflammation into the submucous tissue, with the subsequent ensuing increase of intensity, may be due to the fact that an extensive infection of bacteria has taken place under the influence of the new stimuli, and that their virulence has been intensified. Another source of infection exists in any disease occurring in neighbouring organs, *e. g.*, angina, peritonsillitis, glossitis, parotitis, and angina Ludovici. Laryngitis, following or in association with acute infectious diseases, is probably of haematogenic or lymphogenic origin. The germs in these types of disease are apt to produce suppuration, either by themselves or in company with streptococci and staphylococci (so-called "mixed infection").

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* The term laryngitis submucosa acuta (acute submucous laryngitis) seems to me less suitable than laryngitis phlegmonosa, because not only the submucous tissue, but the mucous membrane, is also affected.

Pathology.—The submucosa is the part most concerned. It is infiltrated by seropurulent or only purulent exudation, which in the latter case leads to abscess. The mucous membrane, where it is inflamed, is swollen, dusky, and very frequently oedematous. This "inflammatory oedema," which must be considered merely as a symptom of submucous or perichondritic inflammation, has been formerly described as a disease sui generis, probably because, as it was the most conspicuous change, it was seen, whereas the deeper process escaped observation. According to the investigations of *Kuttner* and *Felix Semon*, this "inflammatory oedema," which is caused by seropurulent exudation, must be distinguished from the non-inflammatory "congestive" oedema, which is the result of a simple serous transudation.

The "congestive" or "laryngeal oedema" (κατ ἔξοχήν) occurs, just as does the well-known cutaneous oedema in the region of the ankles or eyelids; in all diseases which favour the occurrence of oedema, both generally and locally, *e. g.*, in diseases of the heart, of the kidneys (often as the first symptom), in anaemia and cachectic conditions, and in processes where the venous blood flow in the neck is obstructed, as in goitre, glandular swellings or tumours, aneurisms, and new-growths.

To this class of "congestive" oedema belongs also the oedema observed after prolonged use of iodide of potassium, and the oedema sometimes occurring during the flow of the menses or during the climacteric. *Strübing* has described a sort of *angio-neurotic oedema of the larynx* which manifests itself as a kind of urticaria and is usually associated with a similar eruption on the skin.*

The *phlegmonous inflammation* naturally selects the best conditions afforded for starting and continuing to spread, and therefore tends to occur just where the submucous tissue is normally in greater quantity; or, in other words, where the mucous membrane is only loosely attached to underlying structures. (See p. 356.) The same remark is applicable to "congestive" oedema. We therefore find certain sites of predilection, viz., the anterior (lingual) surface of the epiglottis, the aryepiglottic folds, and the arytenoid cartilages in the first line, and in second line come the ventricular folds and the subglottic region. In former times the oedema occurring at the aditus laryngis was commonly called "*oedema of the glottis*," though actually "*the glottis*," in its old sense, that is, the *rima glottidis, never is the seat of oedema. Hajek* has demonstrated, by injections made on the corpse, that the phlegmonous oedema or submucous infiltration is regulated by the lines of developmental attachment of the submucous tissues. Thus he proved that the oedematous infiltrations of the anterior (lingual) surface of the epiglottis never passes over the free margins of the cartilage into the inside of the larynx, while that of the aryepiglottic fold again is kept back by the pharyngo-epiglottic ligament from spreading in front; so that it quickly extends on to the posterior wall and pyriform sinus.

Subglottic phlegmonous infiltration (phlegmonous subglottic laryngitis) often occurs *independently* of any other disease (in marked contradistinction to *subglottic catarrh*), or in conjunction with an inflammation of the trachea. *Phlegmonous oedema* in submucous inflammation *is usually localised* to the site of inflammation or to the immediately surrounding tissue; but *"congestive" oedema,* on the other hand, is *diffused and not limited to one locality,* and is inclined to spread farther and farther.

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* It might perhaps be doubted whether the oedema following the use of iodine is merely congestive, or whether it might not be of an inflammatory nature, like the coryza also arising from the use of iodine.

Symptoms and Course.—The *symptoms and course differ* according to whether the phlegmonous inflammation is *diffuse,* or whether it is *locally defined.*

(1) *Circumscribed phlegmonous inflammation* may subside spontaneously, or lead to the formation of an abscess. The epiglottis is the part most commonly affected. (See Figs. 151, 152.) Sometimes it originates or is excited by a slight injury received when eating, and either commences in the base of the tongue, or it spreads from the lingual surface of the epiglottis on to the root of the tongue (glosso-epiglottic phlegmon), or again, on to the aryepiglottic folds. In other cases the aryepiglottic folds are the primary seat of the inflammation, or yet again the region of the arytenoid cartilages, less frequently the ventricular folds, and, least often, the vocal cords.

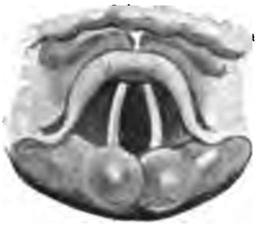

Fig. 151.—"Congestive" oedema of arytenoid cartilages and aryepiglottic folds.

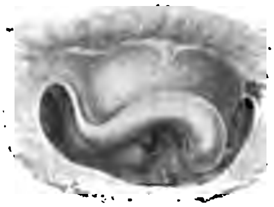

Fig. 152.—Laryngitis phegmonosa circumscripta. Abscess of epiglottis (*Türck*).

The disease starts with fever, pain on swallowing, and hoarseness, and soon causes dyspnoea if it spreads further. By the laryngoscope one is able to note, more or less well defined, a circumscribed redness and swelling, which soon increases and is accompanied by oedema of the adjacent tissue, and leads to abscess formation. If the epiglottis is affected, it appears bright red and enormously swollen. The valleculae and the aryepiglottic folds show oedema, which is manifested by their glassy, translucent, gelatinous, or mucous-polypus-like appearance, and vibration during respiration. The view into the larynx obstructed or rendered impossible. The oedematous pads, is especially those of the aryepiglottic folds, are aspirated into the larynx by the inspired air-current, and aggravate the existing dyspnoea. In some cases the yellowish, translucent pus can be seen bulging. The abscess usually bursts at the margin of the epiglottis or on its lingual surface, or sometimes a little nearer to the tongue. The pus being thus discharged, the inflammation quickly subsides.

(2) *Diffuse Phlegmonous Inflammation.*—The onset of the diffuse phlegmon is more or less acute and violent, and is characterised by pain on swallowing, radiating towards the ear (which, however, in unilateral inflammation need not be excessive), hoarseness, and rapidly increasing dyspnoea. On examination one finds the mucous membrane to a varying extent, on one side only or bilaterally, enormously swollen and red; and later on, perhaps, ulcerated. In subglottic inflammation the same red pads underneath the vocal cords, as in subglottic catarrh, are visible, but are much more swollen and red, and so may considerably narrow the rima glottidis. The affection may here also be limited to one side only.

Erysipelas and the so-called acute phlegmon have been considered as special forms of the diffuse phlegmonous inflammation, as is the case with the pharyngeal phlegmon. It was pronounced *erysipelas* if the inflammation started with high fever and was accompanied by an extensive collateral oedema, but without any tendency to suppuration. On the other hand, it was said to be *acute phlegmon* if, in spite of moderate fever, the general symptoms and prostration were very marked, and if the mucous membrane was more tense and showed purulent infiltration. Both these processes are difficult to distinguish and to differentiate from other phlegmonous affections. One is, however, justified in assuming the presence of erysipelas if other organs, viz., the skin, show erysipelatous disease. Practically speaking, all these fine distinctions are not of any great importance. Phlegmon of the trachea is of rare occurrence, and consists mostly of a direct continuation downwards of a like condition already existing in the larynx.

Diagnosis.—As the clinical symptoms are not very characteristic, diagnosis can only be assured by the use of the mirror. Unfortunately, the laryngeal examination is often rendered impossible because of the swelling in the aditus laryngis; but in some cases pharyngoscopy might help, as it is possible that, by depressing the tongue sufficiently, the epiglottis may become transiently visible, and so admit of an inspection, at least for a few moments. Where, however, laryngoscopy can be performed, it must be done, and it will give us the information we need as to the objective symptoms and their site and extent. Oedema is not to be easily mistaken, for it presents quite characteristic features, viz., the oedematous parts look glassy, transparent, and impart the sensation of a soft but elastic consistency if the probe is applied. More difficult to decide is the question whether there is "inflammatory" or mere "congestive" oedema. In *inflammatory oedema* other signs of inflammation are usually present besides the signs of oedema, whereas *congestive oedema* is distinguished by its pallor and absence of inflammatory symptoms. It is, however, very important, in order to arrive at a definite conclusion, that the whole body should be submitted to a thorough physical examination, especially the heart, kidneys, and lungs.

Prognosis.—Phlegmonous inflammation is always a serious disease, and one which may end in death by suffocation, and in less rapid, but, nevertheless, progressive processes, as septic inflammation within the mediastinum, pleurisy, pneumonia, or general septicaemia. The fatal exitus may sometimes occur quite suddenly and unexpectedly, especially in oedema of the aditus laryngis or in subglottic inflammation.

The *prognosis* is absolutely bad if the primary cause of the inflammation or of the congestive oedema is itself incurable.

Treatment.—The incalculable rapidity of the course of the disease requires careful watching. If there is danger of suffocation, no time must be lost in performing tracheotomy. *Intubation*, which has been suggested, *is contra-indicated in oedema.* (See p. 425.) For the rest, one must consider, if possible, the etiology, *e. g.*, if there is any suspicion of a foreign body, this must be sought for, or an abscess in the neighbourhood must be incised, etc. Treating symptomatically, we may at first try antiphlogistic methods, *i. e.*, ice applications, iced beverages, and crushed ice internally (ice pills), etc.

Menthol pastilles are sometimes very useful in some cases, at least, in the milder ones, and insufflation of renoform powder is worth a trial. Should the patient object to cold applications, then *Priessnitz's* cataplasms are a better substitute, while leeches and mercurial ointment rubbed into the skin over the larynx are of no practical value. The patient must keep absolutely silent. Slight inflammation may subside under this treatment. If an abscess is forming, warm applications and perhaps gargles may be ordered. But if fluctuation has been found, the abscess must be incised without delay by means of a laryngeal knife under the guidance of the mirror. According to *Schrötter*, the epiglottis should be incised from above downwards, while the arytenoid

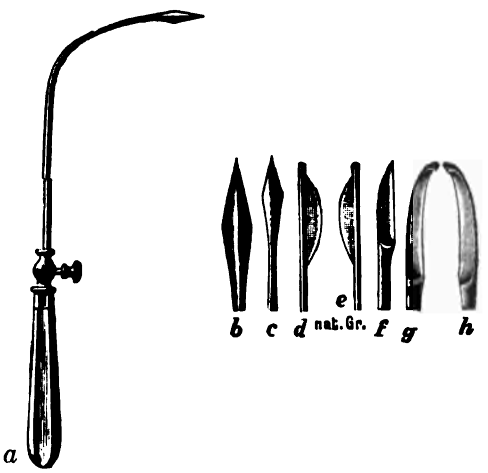

Fig. 153.—Unguarded laryngeal knives (after *B. Fränkel*): *a*, Lancet-knife ,with movable handle (scarificator); *b*, long lancet-knife; *c*, short lancet-knife; *d*, right cutting knife; *e*, left cutting knife; *f*, pointed knife, cutting forwards; *g*, buttoned knife, cutting forwards; *h*, buttoned knife, cutting backwards.

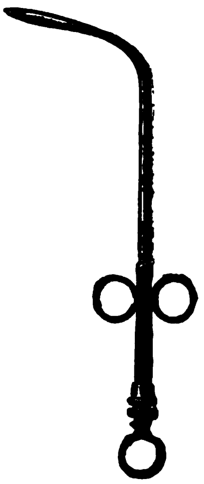

Fig. 154.—Guarded laryngeal knife (after *Tobold*).

cartilage should be punctured straight ahead and the puncture dilated backwards or laterally towards the aryepiglottic folds. If the swelling does not at once subside, the oedematous parts must be scarified with a laryngeal scarificator, after previous local anaesthesia. In cases of urgency a long bistoury may be used whose edge has been guarded to ½ cm. from the point with plaster. (See Figs. 153, 154.)

The "congestive" oedema, which we have discussed here only for the sake of convenience, must be treated on the same lines as "inflammatory" oedema. But it is very important to attend, secundum artem, to the primary disease, as the case may be, which has given rise to the "congestive" oedemå. It is very necessary that the bowels be made to act properly, and we will find it useful to help that action by salines ("bitter salt"). In any case the condition of the heart demands our greatest attention.

3. EXUDATIVE LARYNGITIS.

Exudative inflammations of the larynx are altogether of rare occurrence, and if they occur, they are usually continuous or in conjunction with those of the oral and pharyngeal mucous membrane. As has been discussed (see pp. 186 and 272), it consists of a separation of the epithelium in form of larger or small-sized vesicles, the contents of which are either serous or purulent, and, still rarer, are haemorrhagic. Similar to the eruptions on other mucous membranes, and in contradistinction to those on the skin, these vesicles show very little persistence, and burst so soon that one does not usually see them in their first (unbroken) stage; but they come under our observation as erosions or small ulcers after bursting. Unique cases have been reported in which the laryngeal affection, *e. g.*, herpes, pemphigus, etc., has occurred in the larynx only, or was a primary disease of the larynx.*

In herpes and pemphigus the eruption very often remains localised on the epiglottis or arytenoid cartilages, and at other times the entire laryngeal mucous membrane might be affected.

Symptoms.—Apart from general symptoms corresponding to the seat of the eruption, there are hoarseness, dysphagia, and pain on swallowing; sometimes, however, all the symptoms are very mild, and only a sensation of soreness is complained of. Dyspnoea might be present in a slight or more severe degree.

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* Fibrinous inflammation and pseudomembranous (diphtheritic) conditions will be discussed later on.

Diagnosis.—Owing to the transient nature of the affection, diagnosis is often difficult. In many cases severe catarrhal or even phlegmonous laryngitis,—and if patches, or pseudomembranes are present which unite together,—diphtheria, syphilis, or the effect of caustics may be simulated. We should not fail, however, to make a diagnosis by noticing the presence of vesicles or analogous eruptions on the skin or other mucous membranes; and by its course also we will be able to come to a correct conclusion.

Treatment.—Treatment can only be symptomatic, and will be almost the same as in the analogous diseases of the mouth and pharynx. If the soreness is very unpleasant, menthol pastilles and inhalations of menthol oil may be ordered. Should a simple steam inhaler be at hand, a vapourisation of peppermint oil may be prescribed:

℞.	Ol. menth. pip.	2.00
	Spirit. vini	20.00

F. M. Sig.—Twenty-five drops to the pint of hot water for inhalation.

In special cases painting the affected parts with weak solutions (1 to 2 per cent.) of silver nitrate may be tried. The voice must be rested and spared as much as possible. In pemphigus arsenic has proved very beneficial.

III. CHRONIC LARYNGITIS.

Etiology.—In the majority of cases chronic laryngitis is associated with chronic rhinitis and pharyngitis. The symptoms caused, however, by the chronic laryngeal catarrh are often so marked and predominating, and are so much more complained of by the patient, owing to the greater discomfort of the laryngeal affections, that the simultaneous catarrh of the nose and pharynx are, for the most part, overlooked. If we take into consideration the anatomical and pathological relations, we can easily see that the same irritant agents which cause the nose and the pharynx to become diseased will also act upon the larynx. The etiology of chronic laryngeal catarrh is, therefore, identical with that of chronic rhinitis or pharyngitis. As in the two latter, so also does a *chronic* laryngitis arise out of an acute catarrh if the latter has had no previous opportunity to get well. On the other hand, chronic catarrh comes under our notice, even though an acute stage was not previously noted, mostly as the consequence of chronic congestion of the mucous membrane, if it has been constantly exposed to irritation, no matter how trivial. Professional use or abuse of the voice is one of the most frequent causes of chronic laryngeal catarrh. From the foregoing it may easily be understood why men, especially those of middle age, are more subject to laryngeal catarrh.

We should like here to say again that the secretions from nasal and pharyngeal disease, flowing backwards, often run down into the larynx, where they keep up a sort of chronic irritation of the lower air-passages. This is fostered by the circumstance that in chronic nasal and pharyngeal catarrh mouth-breathing is very common. Chronic infectious diseases, such as tuberculosis, syphilis, and congestive (plethoric) conditions, aggravate the predisposition to chronic laryngitis.

Pathology.—Characteristic of chronic inflammations are the hypertrophy and hyperplasia of the tissues, which are either diffuse, *i. e.*, common to all parts of the mucous membrane, or occur only in certain portions. In some cases the vessels are chiefly concerned: they are dilated and varicose, and especially so on the epiglottis (phlebectasia laryngis). In other cases the epithelium is more affected, and is thickened, scattered over with excrescences, and shows warty or irregular growths (pachydermia laryngis). *Pachydermia* is mostly seen in the tracts of stratified epithelium, viz., on the inner surface of the arytenoid cartilages, on the vocal cords, and on the posterior laryngeal wall; but even the ciliated epithelium may also be affected, and assume an epidermoid character as the result of metaplasia. The mucosa, and if the process be more advanced the submucosa also, is infiltrated with round cells, and the connective tissue, at one place or another, is augmented in quality and quantity. Thus the inflammatory hyperplastic process, more especially in the region of the much-abused vocal cords, leads in due course to a series of changes which must be distinguished according to their site and appearance, but yet are of the same nature, as we have seen. These changes are:

(1) *Chorditis tuberosa* (tuberous inflammation of the vocal cords, also called *trachoma* of the vocal cords), is characterised by the lumpy or tuberous surface of the vocal cords.

(2) *Chorditis nodosa* (nodular inflammation of the vocal cords), the so-called "*singer's nodes*," is seated on one or both edges of the vocal cords, and is produced by a circumscribed hyperplasia of the epithelium and subepithelial layers.

(3) *Chorditis hypertrophica superior* (upper hypertrophic inflammation of the vocal cords), starting originally from a hypertrophic catarrh of the ventricular folds, and spreading on to the upper surface of the vocal cords, leading to the formation of pads and polypi projecting into the larynx. This condition is also called "*prolapsus ventriculi Morgagni*," because it simulates a bulging out of the sinus.

(4) *Chorditis hypertrophica inferior* (lower hypertrophic inflammation of the vocal cords; also called *chronic subglottic laryngitis*), which is characterised by hyperplastic enlargement of the subglottic region. All these four forms, as well as *pachydermia laryngis*, represent the results of chronic inflammation, and are to be considered as varieties of chronic inflammation although the "*singer's nodes*" and the ventricular enlargements show the appearance of new-growths clinically, and the subglottic thickening is described by some authors as a form of scleroma.

As in acute catarrh, so also in chronic catarrh, the muscles or groups of muscles become affected, and this manifests itself by paresis or paralysis of the special muscular functions.

The secretions differ in quality and quantity. If the secretion is, for the most part, purulent, the catarrh is then spoken of as being a *chronic blennorrhea* (*Störk*); but it may likewise perhaps be of a scleromatous nature. Exsiccation and thickening of the secretion, and formation of crusts and scabs, are the features of *dry catarrh* (laryngitis sicca). The secretions may be derived from the larynx or trachea; and the mucous membrane of these parts of the air-passage then shows the same alterations and changes as are found in the mucous membrane of the nose and pharynx in atrophic rhinopharyngitis, foetid or not foetid (see Parts I and III, pp. 75 and 194). It is, however, not yet proved whether the laryngeal mucous membrane becomes actually atrophic, but having regard to its appearance, one would be inclined to assume that it was. Occasionally, cases of "*laryngeal ozaena*" have been described. In long-standing catarrh the vocal cords and ventricular folds may indeed become thinner, so that the interval between them (the opening into *Morgagni's* sinus) might gape widely; and in this case the epiglottis and aryepiglottic folds may likewise become attenuated and thinner. It may, however, be doubted if this is really the result of a chronic catarrh tending to atrophy; or whether all these thinned parts have not been actually thinner than normal, and less developed from the very beginning. There are many persons, quite healthy, and showing neither signs nor symptoms of catarrh, who, however, present a very thin and small epiglottis and aryepiglottic folds.

According to *M. Schmidt*, there is a certain connection between *dry catarrh* and *pachydermia*. The thickening of the stratified epithelium of the posterior wall and of the arytenoid cartilages is due to the constant irritation caused by the dry secretions and the straining as the result of frequent coughing. *M. Schmidt* has observed *pachydermia*, which he aptly terms "corns of the laryngeal mucous membrane," but almost only in long-standing rhinopharyngitis.

Erosions do not often occur in chronic laryngitis, and if ulcers are found, it nearly always means tuberculosis or syphilis, though this might be difficult to prove in any given case.

Symptoms and Course.—The subjective symptoms are more pronounced in the *dry* than in the *secretory* form. Generally, there are complaints of dryness, irritation, tickling or burning in the larynx, which becomes still more marked during speech and singing, and gives rise to frequent cough. If the pharynx is also affected, the discomfort is increased.

The patients do not mind so much the alteration of the voice, provided they are not persons who, by their profession, have to live by their voices. Disturbances of the voice are usually milder in the chronic than in the acute catarrh, but, naturally, the scantier the secretions, the greater the tendency to exsiccation and the formation of scabs, the more the voice will be altered, even to the extent of complete aphonia. But setting this aside, it may be said that the voice will suffer in greater proportion the more pronounced the pathological changes of the vocal cords and the more the vocal cords have lost their capability of normal vibration. *Diffuse*, or still more so "*trachomatous*," thickening of the vocal cords and extensive *pachydermia* are all much more apt to alter the voice than the so-called "singer's nodes," which latter, however, disturb the voice only when speaking softly or singing, because it is easy, by a certain amount of straining, to overcome the slight hindrance and so to close the rima glottidis.

It has been already pointed out that inefficiency of single muscles or groups of muscles must alter the voice; and this is probably one of the chief causes of hoarseness in acute or chronic catarrh. The voice, in the morning especially, is very raucous, as during the night the secretions have accumulated in the larynx, and relief is only obtained when the mucus has been brought up by coughing or after gargling with warm gargles or drinking hot fluids.

Laryngoscopic examination shows a series of pictures exactly corresponding to the anatomical changes. In the mild cases, only insignificantly abnormal signs can be discovered, and a very strong light is required in some cases in order to discover any alterations at all in the colour of the vocal cords, or an isolated swelling or redness in the region of the arytenoid cartilages, or a thickening of the posterior laryngeal wall.

In other cases it is just this thickened ridge on the posterior laryngeal wall which is most conspicuous, and it may be so to such a degree that the adduction of the vocal cords may be impeded, or that, on the attempt to close the rima glottidis (phonation), they become folded into numerous little pleats, which appear almost as if they were excrescences. But that they are not excrescences of the posterior wall can be seen by the fact that these pleats unfold and become smooth during deep inspiration. Occasionally, an erosion might be discovered on the posterior wall, which, as we have said, is especially exposed to mechanical irritation. It is such an erosion which corresponds to the "*rupture of the mucous membrane*" (*Störk*). I, myself, have only twice seen anything which could be interpreted as such a "*fissura mucosæ.*" The results of chronic laryngitis, mentioned above, require a separate description of their respective laryngoscopic pictures.

(1) *Pachydermia laryngis* is often limited to the hindermost part of the margins of the vocal cords. One then sees, on the one side, a rounded, oval or irregular, a wart-shaped or cushion-shaped *projection* of a greyish or greyish-white colour; and, on the other side, the corresponding depression on the thickened vocal cord.

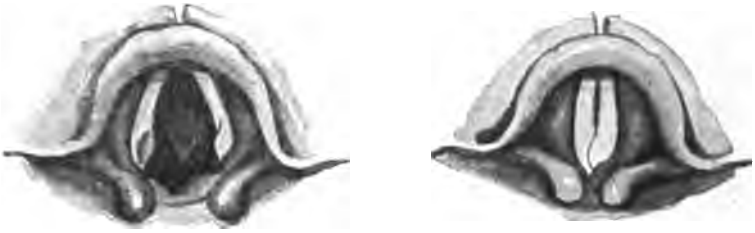

Fig. 155.—Pachydermia laryngis. Thickening of the epithelium of the posterior end of the vocal cords. Pad-like projection of the posterior wall, extending to the left ventricular fold—(*a*) on respiration; (*b*) on phonation.

One almost has the impression, for the sake of comparison, as if a little plate had been stuck on to the vocal process. In other circumstances the thickening extends more forward, but oftener on to the posterior wall (see Fig. 155). The hard and brittle epithelium is very liable to become fissured and eroded. In very marked cases one may be able to see a projection, cracked, fissured, or villous, which looks at the first glance like a tumour.

(2) "*Singer's Nodes.*"—These are protuberances of the size of a pin's head, round or somewhat triangular in shape, nearly always seated symmetrically on the junction of the anterior and middle third of the vocal cords themselves, and are of the same colour. (See Fig. 156.) Usually, one only notices a spindle-shaped thickening of the edge of the vocal cords, which apparently does not partake much of the nature of a hyper-

plasia, for it disappears on closure of the glottis. The "nodes," however, prevent the accurate closure of the vocal cords, more especially if they occur on both sides, and thus cause dysphonia or diphthonia. (See p. 449.)

A similar thickening of the vocal cords—nodular or spindle-shaped—is not infrequently found in children. I have observed it in several cases of children, who, so to speak, have overstrained their voices from their first breath. (See p. 402.) The children are often or are constantly hoarse, and cause a lot of anxiety to their parents, although the prognosis is not unfavourable. Painting the "nodes" is of no value, a fact of which I have thoroughly assured myself. It is best in children to avoid all local treatment and to exhort the parents to trust in and to look forward to the future. With advancing age the differences will become equalised, and so the disorder of the voice will disappear.

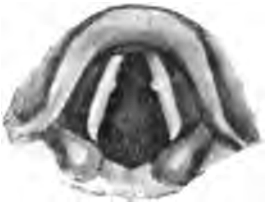

Fig. 156.—Chorditis nodosa (singer's nodes).

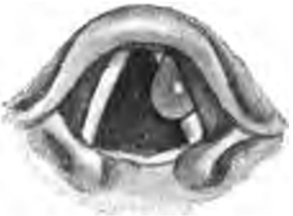

Fig. 157.—Chorditis hypertrophica superior (prolapsus ventriculi *Morgagni*).

(3) *Prolapsus ventriculi Morgagni* must not be misinterpreted. That it does not consist of a bulging out of the ventricle, but that it is really a hyperplastic thickening of the mucous membrane which projects into the lumen of the larynx, has already been said. The vocal cords are sometimes concealed by the red and oedematous ventricular pads if the affection is bilateral. On inspiration the pads recede a little, *i. e.*, they taper slightly; while on phonation they increase and become more prominent. It is, moreover, possible to replace them by means of the probe. Occasionally they are oedematous and then have a glassy appearance and may show vibratory movements.

(4) *Laryngitis subglottica chronica* (inferior or subglottic hypertrophic laryngitis) shows similar appearances to the acute

subglottic laryngitis (due to catarrh or phlegmonous inflammation) (see Fig. 150), with the difference that the stenosis of the glottis produced by the pads projecting from below is not so great as in the acute disease. In some cases subglottic thickening is associated with pachydermia of the vocal cords.

The laryngoscopic picture in dry catarrh need not be separately described, and it only suffices to refer to the pathological changes discussed above (see p. 451).

Diagnosis.—Owing to the circumstance that the clinical symptoms are very often indefinite, diagnosis must be made by means of the laryngoscope. But one should not fall into the habit of declaring every "discolouration" or every "dilated vein" to be a chronic catarrh, even if the patient complains of laryngeal discomfort. The patient's—it has been repeatedly emphasised—capability of referring or localising his sensations is uncertain, and all the troubles complained of by the patient might have their source in another neighbouring organ; while the larynx itself may be quite sound save for the slight "discolouration" or a "dilated vein." On the other hand, we should not be satisfied with the diagnosis of "chronic catarrh" alone, but should aim at ascertaining whether the catarrh is independent of any other disease, or whether tuberculosis or syphilis, etc., is the primary cause. The distinction of a pachydermic thickening of the posterior wall from a tubercular infiltration will sometimes be difficult, more so, particularly, if the thickening keeps within moderate limits. In such a case, if anaemia of the mucous membrane be present, viz., if the mucous membrane is pale, and if there is paraesthesia (see p. 331), our suspicion of tuberculosis must be excited, and we should, therefore, never forget to examine the lungs. In some cases *Killian's* method of examination of the posterior wall will show us an ulcer behind the supposed pachydermia, which at once gives another aspect to the matter, and shows the tuberculous nature of the disease. A probationary excision of some portion will need to be considered only in a severe villous hypertrophy which prevents every function. Microscopic examination, however, if bacilli, or giant-cells, or miliary tubercles cannot be found, will prove nothing. Here only the course of the disease will clear away our doubts.

So also is the distinction between laryngeal carcinoma and pachydermia—not always a certain decision if one has only excised a small portion for microscopic examination. In *pachydermia* the epithelium is sometimes very much thickened, and conical processes are found growing into the deeper tissues, and which, under the microscope, may very closely resemble the "epithelial pearls" ("cell nests") found in carcinoma. It is, therefore, of great importance to remember *that pachydermia mostly, and carcinoma never, occurs on the vocal processes.*

Prognosis.—If we set aside certain forms of subglottic inflammation in children, and especially serious exacerbations, the prognosis quoad vitam is favourable; quoad restitutionem, however, leaves much, if not everything, to be desired. *Nothnagel* maintains that a catarrh of one month's duration is seldom amenable to complete cure. Mild cases, it is true, may be cured, but, unfortunately, they frequently relapse. There are many patients, however, who become accustomed to the continual discomfort, and so resigned to it. But there are others who become real hypochondriacs. Persons again who have to live on the "gold of their voice" have much to put up with from their sufferings. Every treatment, unfortunately, is hampered by the impossibility of removing them from their surroundings or of even merely mitigating them. The prognosis is still more unfavourable if pachydermia or muscular pareses have been added to a long-standing catarrh.

Treatment.—Above everything, the etiology must be taken into consideration. All harmful agencies ought to be eliminated, though it is not necessary to go so far as in acute catarrh. Existing affections of the naso-pharyngeal tract, which are likely to maintain a laryngeal catarrh, must be treated. For the rest, one should abstain from too severe prescriptions, so as not to produce a hypochondriac out of the patient, but to try to adapt to the patient's conditions the hygienic-dietetic measures which should tend to spare and rest the larynx. Naturally, singing, speaking, drinking, smoking, and remaining in smoky or dusty atmospheres should be forbidden, or at least prevented to the utmost, especially in the cases of persons who have to use their voice more than usual, as, for instance, in actors, singers, preachers, teachers, etc.

If we have ascertained that the chronic catarrh is only a part of a general congestive disposition, cathartics, such as purgative salines, are very useful, or a "drink-cure" at Kissingen, Marienbad, Heustrich, and Weilbach may be advised. In addition, alkaline, alkalimuriatic, and saline springs prove very beneficial to catarrh of the upper air-passages. Under certain conditions these mineral waters might also be taken at home, but in any case the "cure at the springs" is always preferable on account of the entire change and alteration of the habits of life. The abstention from all the agents harmful to the larynx, which is assured in most watering-places, helps the therapeutic action of the spring; and, indeed, is often the sole factor. If one looks at the hygienic measures as regards the alteration of the mode of life, having regard to their true value, one will soon come to a decision whether it is preferable to send patients to the mountains or to the seaside. If the patient has the opportunity of moving about in the open air and of resting his larynx, then it is not so important where he goes to. As an exception therefrom are the dry catarrhs, which generally do better in a somewhat humid, *i. e.*, seaside, climate.

The central pivot of all treatment, in the bulk of cases, lies, however, in *local treatment*. Here astringents are most useful. I use silver nitrate (2 to 10 per cent.) for painting in such a manner that, according to the irritability of the mucous membrane, I paint every two or three days and gradually increase the concentration of the solution, while adapting the treatment to each particular case. In dry catarrh inhalations are prescribed, in order to loosen the secretion, and afterwards the mucous membrane is then painted. As in pharyngitis (see p. 253), so also in dry laryngitis, iodine is very useful and may be given internally and externally. Towards the end of the treatment the intervals between the paintings are to be lengthened.

Circumscribed thickenings or erosions can be cauterised (under local anaesthesia) with lapis infernalis, chromic acid, etc.; and for this purpose a suitably bent and guarded caustophore must be used.

Treatment of sequelae is indicated only where special complaints of discomfort are made; otherwise their treatment is identical with, and is contained in, that suitable for chronic catarrh.

Pachydermia may be treated in the manner above described by cauterisation (lapis, chromic acid, trichloracetic acid). If this fails, it will become necessary to proceed on surgical lines, if the subjective symptoms are very distressing (pain on swallowing, dyspnoea, alteration of the voice). The pads or the thickenings may be excised by means of a double curette, made to cut vertically. (See Fig. 158.)

After the operation, it is absolutely necessary that the patient should rest his voice completely for some weeks at least.

"*Singer's nodes*," if large enough to cause discomfort, as in actors or singers, may best be removed by means of a sharp scooped forceps. I prefer *Rosenberg's* instrument, which can be fixed to *Krause's* universal handle (see Fig. 159), and follow *M. Schmidt's* instructions, who only snips off the node if it cannot be easily excised. Thus any unintentional injury may be avoided, viz., a pinching off of the mucous membrane of the vocal cords. I consider galvano-cauterisation most unsuitable, if not even harmful. Here also silence must be preserved after the operation, until every sign of reaction has subsided. In some cases such a "silent cure," in combination

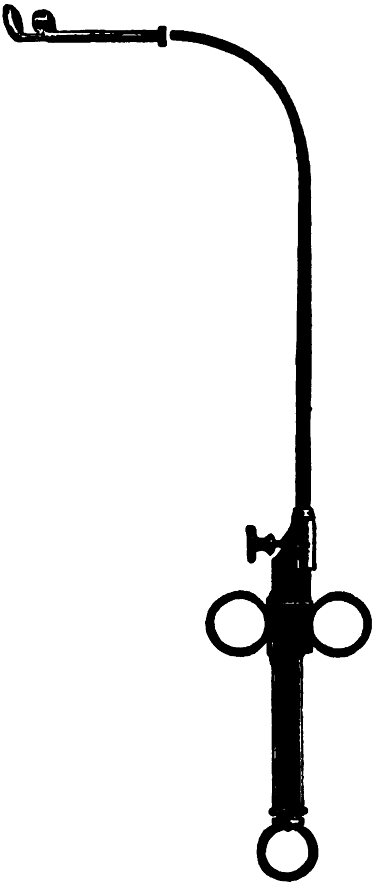

Fig. 158.—Double curette cutting vertically (after *Landgraf-Krause*).

with painting with silver nitrate, is sufficient to remove small prominences; and anyhow it is worth a trial. In children such "nodes" should be let alone.

Prolapsus ventriculi and the subglottic pads may also be operated on by means of *Rosenberg's* scooped forceps or a small double curette. In threatened suffocation from subglottic swelling, tracheotomy or intubation, as recommended by *Schrötter* or *O'Dwyer*, might be necessary (*cf.* with what is said of treatment in scleroma).

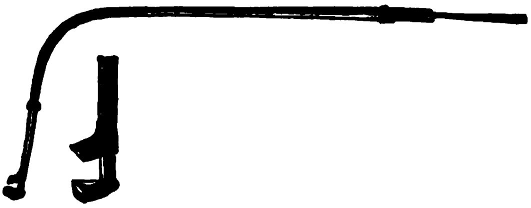

Fig. 159.—Scooped forceps (after *Rosenberg*).

IV. PERICHONDRITIS.

Etiology.—The cause of laryngeal perichondritis, inflammation of the perichondrium, as of phlegmonous laryngitis, is a bacterial infection, mostly, if not exclusively, due to staphylococci and streptococci immigrated from the surface. But simple laryngitis also is finally brought about by micro-organisms. As to why, in the one case, only the superficial layers, and in another case the submucous tissue, and in yet another the perichondrium, become affected, it would be difficult to explain. Possibly here, as elsewhere, general or local anatomical conditions, and the nature and virulence of the micro-organisms, contribute towards the decisive result. How the infection takes place in a certain number of cases is not easy to say. These particular instances have been grouped together and termed cases of idiopathic or primary perichondritis, and, for want of another or better explanation, they were traced to a specially severe "chill" or to overstraining the voice. In face of this somewhat obscure view we must adhere to our opinion, and again emphasise the statement, that perichondritis is caused by micrococcal infection. It would be better to altogether give up the old traditional classification of idiopathic (primary) and symptomatic (secondary) perichondritis, and to speak simply of *perichondritis.*

In most cases the gate of entrance for the bacteria is not difficult to discover. It is chiefly in diseases which are accompanied by or lead to ulceration, and, above all others, tuberculosis and syphilis, where the ulceration is the intermediary of secondary infection. In a similar way perichondritis can be explained, which sometimes arises from an injury or foreign body in the larynx. Naturally, the deeper the loss of substance is situated, the easier it is for the perichondrium to become denuded, and thus affected. Associated with these changes in the cartilaginous skeleton is always an inflammatory oedema of the soft parts of the affected organ or its adjacent tissues. Oedema and perichondritis, therefore, as we have seen, are concomitant occurrences; the oedema is the process farthest advanced on the surface of the mucous membrane, and the perichondritis is the affection which has spread deepest into the underlying tissues.

Less frequently than from surface lesions, infection occurs by the way of the blood- or lymph-vessels. Such a metastatic perichondritis is occasionally met with in pyaemia, acute polyarthritis, typhoid, etc.

Pathology.—Infection by bacteria leads to inflammation of the perichondrium and subsequently to suppuration between it and the cartilage. The perichondrium is separated from the cartilage and bulges forwards; but as the cartilage is then bereft of its blood-supply, it must become subject to necrosis to a greater or less extent. In the case of *internal (laryngeal) perichondritis,* the abscess projects towards the lumen (laryngeal side) of the larynx; in *external perichondritis,* viz., in an affection of the external perichondrium, suppuration tends outward through the skin. There are also cases where the pus burrows through from the one side to the other (from within outwards or vice versâ).

The *bursting of the abscess towards the inside usually* takes place soon if it was formed as the result of an ulcer, and in this case there is not much projection; otherwise the bulging might be very considerable before it opens.

The site of bursting depends, of course, on the seat of the abscess. Necrosed cartilage can often be seen projecting from the abscess cavity, and if it is separated, it will be discharged by coughing; and it is even possible, in some cases, to recognise the origin of the cartilage from its shape. In other cases the loosened cartilage may be caught between the rima glottidis and cause severe dyspnoea or even death from suffocation. The same danger of suffocation might be threatened if the cartilages are necrosed to a very great extent, and the larynx, after the discharge of the necrosed parts, tends to collapse.

In external perichondritis the resistance of the parts covering the outside of the larynx may be sometimes so great that the pus does not find a direct pathway outwards, but tends to form a migrating abscess.

In *bilateral perichondritis*, if the pus bursts through the skin, as well as into the lumen of the larynx, *a complete fistula* will be established. Such a laryngeal fistula, as a rule, shows a tortuous course, owing to the fact that the outer perforation is usually on a lower level than the inner opening.

Symptoms.—Perichondritis is mostly limited to one cartilage only. Most commonly it is the arytenoid cartilage which is affected, for it is also most frequently the seat of ulceration. After it comes the cricoid cartilage, and, not infrequently, in conjunction with the arytenoid. The thyroid cartilage also, and least often the epiglottis, may be the seat of perichondritis. Hence the objective symptoms differ according to the seat of the disease.

(1) *Perichondritis Arytenoidea.*—The disease is more frequent on one side only than bilaterally. (See Fig. 160.) Laryngoscopically, one sees an oedematous swelling in the region of the arytenoid, covering sometimes in excessive cases a portion of the vocal cord, and thus concealing more or less an ulcer which might happen to be present. The oedema spreads into the aryepiglottic fold. Mobility of the vocal cord is restricted or entirely abolished. Necrosed cartilage may be visible, and then a small quantity of pus appears, especially if gentle pressure be made from the outside.

(2) *Perichondritis Cricoidea.*—This very often has its origin in

ulcers on the posterior laryngeal wall, for example, from tuberculosis or typhoid. (See Fig. 161.) If, as is often the case, the cricoid plate and the arytenoid cartilage are simultaneously affected, the mirror shows a swelling over the region of the arytenoid and also over the posterior wall to a varying extent, sometimes right down to the subglottic space. If the external perichondrium of the cricoid is affected, the swelling can be seen in the pharynx and pyriform sinus. In the case of the anterior arch of the cricoid being diseased, the anterior region of the neck is prominent and very tender. If the seat of the disease is in the region of the crico-arytenoid joint, then mobility of the corresponding vocal cord is materially inhibited.

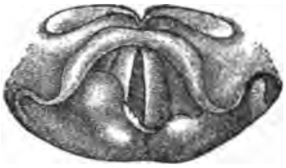

Fig. 160.—Right-sided perichondritis arytenoidea (*Türck*). The swelling of the arytenoid cartilage and aryepiglottic fold of the right side covers a tubercular ulcer, leaving only a small area of it visible.

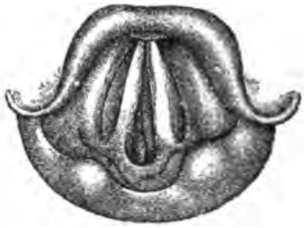

Fig. 161.—Left-sided perichondritis cricoidea (*Türck*). On the left side is considerable subglottic swelling, corresponding to the perichondritis of the cricoid; the subglottic swelling is seen spreading on to the posterior laryngeal wall. Oedematous swelling of left arytenoid and left aryepiglottic fold. The glottis is greatly narrowed.

(3) *Perichondritis Thyroidea.*—If the external surface only is affected, a unilateral or bilateral, soft and very tender swelling may be noticed in the front of the neck. Inflammation of the laryngeal perichondrium shows itself, according to its seat, as a swelling in the region of the anterior commissure or on one side only. In the latter case the swelling might be so considerable that the ventricular fold, ventricle, and vocal cord are each hardly distinguishable.

(4) *Perichondritis Epiglottica.*—The perichondrium of the epiglottis is so firmly adherent to the cartilage that it cannot be separated from it by a collection of pus. It so happens rather

that the cartilage itself is eroded and necrosed by tubercular, and still more frequently by syphilitic, ulceration. It is possible in such a case to see the irregular edges of the eroded cartilage. The adjacent structures, of course, also show here marked oedematous swelling.

SYMPTOMS.

The subjective symptoms of perichondritis are not very characteristic, as there may be hoarseness, which, however, in external perichondritis might be altogether absent, pain on swallowing, and dyspnoea. Hoarseness is often caused by the primary disease (tuberculosis, syphilis, etc.), or it may be due to a mechanical hindrance of the mobility of the vocal cords or to the muscles being implicated. The pain on swallowing frequently radiates towards the ear, and may be agonising, especially if the arytenoid, cricoid, and epiglottis are affected. External pressure upon the diseased part is very painful, and it is just this localised tenderness which will often guide us as to the correct seat of the disease. Dyspnoea is the greater the narrower the lumen is rendered by the swelling, and it may assume the most dangerous degree in consequence of an abscess having burst into the larynx, or a necrosed part of a cartilage being caught in the glottis (see above), or, by the fixing of the vocal cords in the attitude of approximation.

The course is either acute or chronic, according to the underlying disease, *e. g.*, acute in typhoid or after injury, chronic in the ulcerative processes of a tuberculous, syphilitic, or carcinomatous nature. In the latter case it not infrequently takes some time before the cartilage is separated and discharged, and the pus which runs down into the lower air-passages may cause gangrene of the lung. In all these cases intercurrent complications may happen and aggravate the preëxisting danger, or even cause death from suffocation. In very favourable cases the necrosed cartilages are discharged, and recovery may take place with the formation of scars, leading to immobility of the cartilages and stenosis.

Diagnosis.—Internal perichondritis cannot be diagnosed with certainty unless one is able to see or touches with a probe necrosed cartilage, or if such a part has been coughed up. On the other hand, arytenoid perichondritis, for instance, can easily be mistaken for an infiltration over the arytenoid cartilage, particularly at the commencement of the disease. The subjective symptoms are of little value if the primary cause, *i. e.*, the underlying disease, has already produced various disturbances. We may, however, assume, with a certain degree of probability, that we have to deal with an arytenoid perichondritis, if, following on a sloughing process, fever and pain, perhaps also dyspnoea, are accompanied by a swelling in the region of the arytenoid cartilage, and the corresponding vocal cord is impeded in its movements. If a perichondritic abscess has burst into the larynx, we sometimes see by the laryngoscope that pus is oozing from a certain spot if we exercise a little pressure from outside. At the same time there is a conspicuously isolated tenderness on pressure from outside or on touching the spot with the probe from inside. Such tenderness, however, is not pathognomonic of perichondritis, but can also be found in inflammation of the crico-arytenoid joint.

External perichondritis may be confused with suppurating lymphatic glands, new-growths, or gummata.

It is very important, but also very difficult, in a given case to ascertain the primary cause of the perichondritis. In doubtful cases, especially if hoarseness has previously existed for some time, tuberculosis and syphilis should be borne in mind, for these diseases are the most frequent causes of perichondritis.

Prognosis.—The manifold complications and dangers which sooner or later occur make the prognosis very serious. It is absolutely unfavourable in tuberculosis or carcinoma, a little better in syphilis, and even in the mildest cases severe functional disturbances are the rule.

Treatment.—Causative treatment can only be considered in cases of syphilis where an energetic antisyphilitic treatment may be applied. On the other hand, symptomatic treatment may do some good, though within moderate limits. At the commencement the larynx must be absolutely rested, and ice applied externally and internally. Soon, however, the ice must be replaced by warm fomentations. If one has found fluctuation, the abscess must be incised by means of a guarded laryngeal knife. A cartilaginous sequestrum should be removed, *i. e.*, extracted by means of a forceps if it is movable. Great attention must be directed to respiration, and if this is threatened, tracheotomy should be performed without delay. For the rest, one may try to alleviate the suffering by the instillation of menthol oil, 20 to 50 per cent. cocaine, adrenalin 1 : 1000, or narcotics. Ultimately stenosis, which might result, must be treated by the methods previously described. (See p. 408, et seq.)

V. DISEASES OF THE JOINTS.

Etiology.—Until recently diseases of the laryngeal joints have not often been made the subject of exact investigation, and in some text-books are not even mentioned. Whether they are of rare occurrence may be doubted, though some authors incline to that opinion. This neglect may be partially explained by the difficulty met with in interpreting the indefinite symptoms intra vitam, and partially by the lack of sufficient postmortem investigation. This may especially be said of the acute inflammatory processes of the joints, which cause few subjective or objective symptoms, and soon get well. Such inflammations are, perhaps, more frequent than is usually thought, and there is usually some paraesthesia, some indefinite sensation of pain in the region of the larynx, which may finally be traced to disease of a laryngeal joint, just analogous to those slight joint affections as occur in the course of acute rheumatic arthritis.

It is, indeed, the acute polyarthritis which plays a chief rôle in laryngeal joint disease. We leave it open as to whether such a joint disease in the larynx must be considered as being metastatic, as, for instance, in the case of gonorrhoea, where it has also been observed. Much could be said in support of this view if one conceives the acute polyarthritis as being a mitigated pyaemia. Besides rheumatic and gonorrhoeal arthritis, there are also other infectious diseases which involve the laryngeal joints, *e. g.*, typhoid, diphtheria, variola, tuberculosis, and syphilis. In some of these forms of arthritis the inflammation runs from the outset a somewhat *chronic* course, as it also does in gout and likewise in those cases where a phlegmonous or perichondritic inflammation spreads into the laryngeal joints.

Pathology.—The crico-arytenoid joint is the one most often concerned. It may be affected in the course of a true polyarthritis, together with other joints of the body or *alone*. In some cases the crico-arytenoid arthritis is, so to speak, the forerunner of other joint affections. There is at first a serous or seropurulent exudation into the joint, but not infrequently the peri-articular tissue may also be infiltrated. Inflammation may occur on one or on both sides. If the inflammatory process is of a more chronic form, it finally leads to ankylosis and to a true ankylosis if it is intracapsular, and spurious ankylosis if it is extracapsular. In the latter case the ankylosis is occasioned by cicatricial tissue, fibrous thickening, or adhesions.

Symptoms.—In the mild cases of rheumatic arthritis patients complain of an unpleasant feeling of tension, which is localised in the lateral region of the larynx during speech and swallowing, and this sensation is aggravated on touching the spot with the probe or finger. In more severe cases, however, the pain may be very considerable and radiate towards the ear. It is accompanied by hoarseness and sometimes even by dyspnoea. The pains are more pronounced in the recumbent position. If one exercises a gentle pressure on the upper and back part of the thyroid cartilage, a crackling noise may be heard and felt, which is supposed to be pathognomonic, as *Grünwald* points out. It is heard at the same place which also causes pain on pressure. The crepitation is due to the friction of the inflamed and roughened synovial membranes.

The laryngoscopic examination is not always characteristic, especially if there is nothing but a simple exudation into the joint. In severe cases, on the other hand, reddening and swelling over the arytenoid cartilage may be found; the mobility of the vocal cord is reduced or inhibited, and may sometimes be seen to be quite immobile in the position of respiration or phonation. In bilateral acute or chronic inflammation the picture is more complicated. The vocal cords are then symmetrically fixed in abduction or adduction, but more often asymmetrically, and then an unequal mobility of both cords is most conspicuous. In some cases the arytenoid cartilage seems to be dislocated and lies at a quite unusual angle; at another time it might be possible to see scars and adhesions which would at once permit the recognition of a spurious ankylosis.

Diagnosis.—If one is able to see by the laryngoscope reddening and swelling in the region of the arytenoid cartilage, which is accompanied by immobility, or, at least, a reduction of mobility of the vocal cords, in association with pain on swallowing and pressure, then a peri-arthritis is the next thing to think of, *i. e.*, an infiltration in the neighbourhood of the joint; or a perichondritis should be borne in mind. But if there are other joints of the body affected, it may be assumed, with some degree of certainty, that we have also to deal with an inflammation of the crico-arytenoid joint. If in doubt, await the course of events. In superficial examinations a mistake with hysterical paraesthesia may arise. The differential diagnosis between paralysis of the vocal cord and joint affection is difficult. Acute inflammation might betray itself by its course, the characteristic pain on pressure, and very frequently by the crepitation before mentioned. Still more difficult is the differentiation between chronic arthritis and paralysis. Here one can only arrive at a correct conclusion by carefully weighing the anamnesis and the previous course, together with the laryngoscopic finding, especially if there are scars and adhesions present. In paralysis due to disease of the nerves or muscles, the patients usually never complain of pain. Anyhow, diagnosis in cases which have already run their course is very uncertain.

Prognosis is favourable in the acute joint diseases, especially in rheumatic arthritis, though relapses are frequent, and the condition may even pass into the chronic stage, with resulting ankylosis. If there already exists a chronic inflammation or ankylosis of the joint, prognosis is, to say the least, doubtful. If the vocal cords are fixed in the mesial line or near to it, stenosis, as in typical paralysis of the posticus, may occur.

Treatment.—In acute arthritis the larynx must be completely rested, and ice or *Priessnitz's* cataplasms applied, and aspirin or salicylic acid administered internally. Chronic inflammation, if based on syphilis, must be treated by mercury and iodide of potassium. If it prove necessary, the scars, adhesions, or thickenings which are visible and accessible may be operated on; and stenosis must be dilated after the method previously described. Unfortunately, this is very unsatisfactory. In sudden threatening dyspnoea, luckily not very frequent, tracheotomy should be at once performed.

VI. ACUTE AND CHRONIC INFECTIOUS DISEASES.

1. ACUTE EXANTHEMATA.

Measles.—The larynx and trachea, as well as the pharynx, show, during the first few days of measles, a blotchy redness of the mucous membrane, analogous to the cutaneous rash. Apart from this appearance, the whole respiratory tract is affected, showing symptoms of catarrhal laryngo-tracheitis (hoarseness or other disturbances of voice, coughing, expectoration of mucus, etc.). If the secretion is copious, dyspnoea might ensue, especially if the subglottic region is swollen. If such is the case, it is probably due to a pyogenic infection, which might lead to the formation of an abscess or perichondritis. In other cases, and then mostly in the stage of the fading of the rash and desquamation, real pseudomembranes may be formed, which are evidently caused by secondary infection from diphtheria bacilli.

Scarlet Fever.—The larynx and trachea are much less often affected than the pharynx, but it not infrequently happens that the diphtheria-like scarlatinous angina spreads on to the aditus laryngis, and, as has been observed in various cases, down into the trachea. Nephritis, which occurs so often in scarlet fever, may cause congestive oedema of the larynx.

Small-pox.—Apart from a catarrhal laryngitis, pustules or fibrinous exudations, and phlegmonous inflammation also, may occur in the region of the larynx, leading to perichondritis or abscess.

Diagnosis.—In all the foregoing diseases the diagnosis should offer no difficulties if the whole syndrome of symptoms is taken into consideration, especially having regard to the cutaneous rash.

Treatment.—The treatment is usually symptomatic. Local treatment is nearly always superfluous. In severe cases, however, tracheotomy might become necessary.

2. WHOOPING-COUGH (PERTUSSIS).

Whooping-cough is a neurosis of the upper air-passages caused by some infectious germ not yet ascertained. It is always accompanied by some catarrh and characteristic paroxysms of coughing. Two stages are distinguishable: the first is a simple catarrhal stage, which shows the usual symptoms and signs of an ordinary catarrh of the upper air-passages; this then passes on into the second stage, which is convulsive, and is characterised by the typical spasmodic attacks of paroxysmal coughing.

The coughing paroxysms are especially alarming and more frequent during the night. The short coughs follow each other rapidly in series, and are characterised by intervals of loud inspiratory "whoops." The attacks occasion great cyanosis and congestion, which may lead to nose-bleeding, conjunctival or retinal haemorrhages, or ecchymosis beneath the laryngeal mucous membrane, etc.

The inspiratory stridor is probably due to spasm of the adductors of the vocal cords. The cause of the paroxysms themselves is not yet exactly known, and the laryngoscope gives us no clue. Very often nothing pathological can be seen, and the redness sometimes found in the larynx is probably the result, not the cause, of the coughing. In some severe cases secondary diphtheritic infection and real spasm of the glottis, accompanied by general convulsions, have been observed. We refer the reader, however, to text-books on medicine for fuller information.

Treatment.—This must abstain from all manipulations of the upper air-passages, especially as there are no local changes. Of all the numerous specific drugs, none has proved infallible, as any one will admit who is sufficiently unprejudiced. Morphine and codeine in combination with bromides are likely to mitigate the paroxysms, but are without any effect on the course of the disease. I desire to recommend, however, the use of oil of cypress (oleum cupressi), which has been so much praised by *Bravo* and *Soltmann*. It is best to drop an alcoholic solution of the oil (1 : 5) upon the bolster, pillow, sheets, and body linen of the children. The fluid is used four times a day and once or twice during the night, about ½ ounce in all. The aromatic smell of the oil is usually liked, and the only disadvantages are the yellow stains which remain. How this expensive drug acts is not yet ascertained.

3. INFLUENZA.

The catarrhal form of influenza is almost always associated with laryngo-tracheitis, and shows the desquamation of the epithelium in the region of the vocal cords already described in acute laryngitis. These erosions present themselves as opaque patches which contrast vividly with the bright-red surroundings, and impart to the vocal cords a conspicuously patchy appearance. Various authors ascribe this appearance to a fibrinous exudation. In some cases the process might spread into the deeper tissues and, indeed, might lead to a submucous infiltration, perichondritis, abscess, and paralysis of the posticus.

Symptoms.—The symptoms depend mainly on the seat and extent of the disease, which very often is most resistant and shows great tendency to relapses.

Treatment can only be symptomatic.

4. DIPHTHERIA.

Etiology and Pathology.—Pharyngeal diphtheria not infrequently spreads down into the larynx and trachea, and even beyond this into the bronchi, especially in weak, scrofulous, or tuberculous children (descending diphtheria). Exceptionally, diphtheria begins in the larynx or trachea, or even in the lower regions of the air-passages, and mounts upwards into the pharynx (ascending diphtheria). Sometimes the affection is limited to the larynx and trachea only. The disease is caused by the *Klebs-Löffler* bacillus, whose biological relations have been discussed in the chapter on Pharyngeal Diphtheria. (See p. 300.)

Diphtheria occurs as a secondary complication in various infectious diseases, such as typhoid, measles, whooping-cough, scarlet fever, etc., or in severe cachexias.

Laryngeal diphtheria is sometimes also called "croup." This term was originally used to signify merely a very conspicuous symptom, namely, the rough, loud, raucous ("brassy") cough, and the noisy, sibilant respiration. Later, relying on the authority of *Rokitansky* and *Virchow*, the expression was applied to the pathological anatomical change, *i. e.*, to a process which is characterised by fibrinous exudation upon the surface of the mucous membrane, and to the formation of loose removable membranes, in contradistinction to true diphtheria, where the fibrinous exudation takes place into the epithelium and deeper portions of the mucous membrane, causing it to necrose. According to this difference, "croup" and "diphtheria" must be clinically separated. Careful investigation, however, will show that such a distinction cannot be carried out, either anatomically or clinically. Croup and diphtheria are pathologically identical, and may perhaps be considered only as varying degrees of one and the same disease caused by the *Klebs-Löffler* bacillus. It is perhaps best to entirely drop the term "croup," which has given rise to such clinical and anatomical confusion, and, moreover, to complicate the matter yet more, must be distinguished from "pseudo-croup." (See p. 434, et seq.) In its proper place one should only speak of mild or severe diphtheritic inflammation of the larynx, or shortly, of "*laryngeal diphtheria.*"

True fibrinous exudation on the surface admittedly occurs as the result of mechanical, chemical, and thermic irritations. This so-called *fibrinous laryngitis*, sensu strictiori, is analogous to the fibrinous process sometimes observed in the nose and pharynx. It is usually due to scalding or burning or an unintentional injury during intralaryngeal galvano-caustic operations. Certain micrococci are supposed to play a part in fibrinous laryngitis, and we leave it an open question whether they are the actual cause, or are later secondary importations. It is certain, in any case, that in the larynx, as well as in the nose and pharynx, fibrinous exudations which do not contain diphtheria bacilli are found. Negative findings, however, are no proof, and, practically speaking, one should act wisely if one considered a case of fibrinous laryngitis as suspicious of diphtheria, and should isolate it, that is, if it cannot be traced to a galvano-caustic operation or to other chemical or thermic cause.

Pathology.—The changes met with in laryngeal diphtheria are essentially the same as in the pharyngeal diphtheritic disease. (See p. 300.) As a rule, the diphtheritic process, if it occurs in the larynx, does not spread as much into the deeper tissues as it does in the pharynx, but, on the other hand, it shows a distinct inclination to spread along the surface—at times so much so that actually exact casts of the larynx, trachea, or finer bronchi are formed. Herein also lies the great danger of a *descending diphtheria,* in that all the air-passages may become blocked. The lungs may suffer in two ways: they may be infiltrated, as in pneumonia, by the fibrinous exudation, or they may become atelectatic, because of the blocking of the supplying bronchi, and subsequently to this a vicarious emphysema may develop in other parts of the lung. Very important are also the conditions of the glottis. The pseudomembranes which adhere more firmly to the stratified epithelium than to cylindrical epithelium may here soon stenose the larynx in a dangerous degree, and this danger of suffocation is still more increased by any subglottic swelling. In some cases it is not the formation of pseudomembrane in the glottis alone, but the subglottic and submucous oedematous infiltration, which constitute the real dangers.

Symptoms and Course.—Laryngeal diphtheria usually starts in this way: with and after *certain symptoms,* pharyngeal diphtheria invades the larynx. The symptoms which characterise this invasion are *increase of temperature, hoarseness, "brassy" (raucous) cough,* and *dyspnoea.*

Whenever, in the course of pharyngeal diphtheria, this syndrome of symptoms occurs, a descending diphtheria may be assumed, with the greatest probability as to correctness. I say with all the greatest *probability,* not *certainty;* for severe catarrhal laryngitis or phlegmonous inflammation may also cause the same symptoms, such as hoarseness, dyspnoea, and "brassy" cough.

I desire once again to call attention to the need of bearing in mind subglottic laryngitis. (See p. 435.) One must be in doubt whether it partakes of diphtheria or not, especially in those cases where the symptoms above mentioned are still present, while the pharyngeal diphtheria has already subsided, or at least is hardly any longer recognisable, or if the diphtheria has arisen in the larynx primarily. To the foregoing may be added the fact that in some cases one or other symptoms may be altogether missing, viz., if the glottis and the subglottic region are free. But in all these cases the further course of the disease will soon clear up the matter. Dyspnoea, which perhaps occurs from the first, like a danger-signal, in brief paroxysms, soon increases and bears witness of true laryngeal and tracheal obstruction, accompanied by the characteristic stridor. (See p. 386.)

Pseudomembranes or portions of them are perhaps brought up by an attack of coughing, which then frees the larynx for a while and the child may fall asleep. Soon, however, the attacks of dyspnoea recur, which then strain all the auxiliary muscles of respiration in the neck and thorax to the utmost, thus deepening or lengthening the efforts at respiration. Paroxysms of violent dyspnoea rouse the children, who, in their urgent desire for aid, clutch wildly at their throats. These paroxysms alternate with exhaustion; the voice becomes aphonic; the cough loses its sound; and, finally, the little ones succumb. The respiration becomes rapid and shallow, but seemingly freer; the pulse quick, feeble, and irregular, or even intermits for some moments, and with the signs of profound cyanosis and stupor the child dies from suffocation or exhaustion.

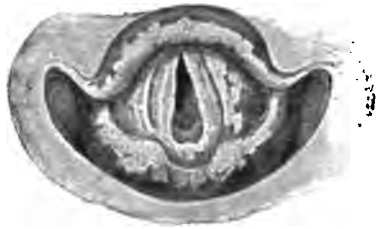

Fig. 162. — Laryngeal diphtheria (*Türck*).

In the rare cases of adults who suffer from diphtheria stenosis does not usually appear before the bronchi have also become involved.

The laryngoscopic picture is a very varying one. At first, patches may be seen on the laryngeal surface of the epiglottis or on its edges, or on and between the arytenoid cartilages, on the ventricular folds, or on the vocal cords. Later on false membranes may be observed, which are often partially separated at one or the other place, especially in adults, and are then seen floating or moving about. At the point where such a pseudo-membrane has been cast off the mucous membrane appears much reddened, and is sometimes seen to be slightly bleeding. The subglottic pads beneath the vocal-cords at times project a great deal into the lumen.

There are cases where the whole process is localised, and remains limited to the larynx. In the great majority of cases, however, white patches or pseudomembranes may be also observed in the trachea during respiration.

If the disease subsides, the pseudomembranes are cast off and expectorated. Owing to its small tendency to penetrate the deeper tissues (see above), restitutio ad integrum usually ensues. On the other hand, if the infiltration was severe, or if ulcers have developed, then scars and adhesions are the consequence, which lead to stenosis on account of their retraction. Post-diphtheritic paralysis in the region of the larynx has been discussed in the chapter on Pharyngeal Diphtheria. (See p. 300.)

Diagnosis.—If laryngeal diphtheria occurs in association with pharyngeal diphtheria, a mistake can hardly be made, even if a laryngoscopic examination cannot be made. But there might arise some difficulty in diagnosis if, as has already been pointed out, on examination of the pharynx nothing can be found characteristic of diphtheria. In that case some items of the history of the case will point to it; or, on the other hand, if the larynx was the primary seat of diphtheria. Should laryngoscopy fail, then a differential diagnosis between the prognostically favourable catarrhal "pseudo-croup" and a phlegmonous laryngitis could be inferred from the clinical appearances. As has been said previously, the onset of "pseudo-croup" is sudden and occurs in previously healthy children or in those who are only slightly ill (see p. 435); while diphtheria manifests itself by distinct premonitory symptoms. The differential diagnosis from phlegmonous laryngitis is sometimes very difficult or even impossible, and often can only be made by observing the course of the disease. At times our attention is directed to diphtheria by other cases occurring simultaneously; or the coughing up of membranes or the presence of *Klebs-Löffler's* bacilli will reveal the nature of the disease. Fibrinous exudations caused by caustics or burning are almost always unaccompanied by fever.

In adults, laryngoscopy, as a rule, is decisive. *Plaut-Vincent's* angina (p. 198) may occasionally spread into the larynx and form membranes which strongly resemble diphtheritic pseudomembranes. Recently *Reiche* has described a case where the *fusiform* bacillary angina was limited to the larynx,—laryngitis ulceromembranosa,—and where no other bacillus than the fusiform kind was found. It is necessary to bear in mind this comparatively benign disease, although its occurrence may be rare.

Prognosis.—Diphtheria is always a serious disease, and the more so if the child has been weakened by a previous illness and has a small larynx. By its descending into the lower air-passages the prognosis becomes worse, and even hopeless, if respiration becomes obstructed. (For the rest see the chapter on Pharyngeal Diphtheria.)

Treatment.—With regard to treatment, more particularly that by injections of serum, we must refer to what has been already said in previous chapters. (See pp. 300, 306, et seq.) Owing to the gravity of the disease, a large dose of serum should be injected at once. The further treatment must be symptomatic and antiphlogistic: ice applications, crushed ice to suck (ice pills), and basins with boiling water may be placed close to the bed, and stimulants and restoratives administered. Local treatment seems superfluous, if not actually injurious.

If stenosis is threatening and is manifesting itself by the inspiratory drawing-in of the lower part of the thorax, then intubation or tracheotomy is required. Generally speaking, in diphtheria, intubation, according to *O'Dwyer's* method, is preferable, provided that one can manage it and trained nurses are at hand. But if asphyxia has already supervened and if the disease shows a marked tendency towards gangrene, or if it has spread further down into the trachea, then only tracheotomy is justifiable. Tracheotomy must likewise be performed if intubation fails or has had just the opposite effect to that which was expected, as, for instance, by the tube having separated pseudomembranes and then having pushed them before it while it was being introduced. (See p. 425.) Unfortunately, there are many cases where tracheotomy fails, either because the diphtheritic process continues to spread downwards below the wound, or because other complications on the part of the heart or lungs frustrate all treatment. Nevertheless, the favourable influence of the serum, on both intubated or tracheotomised children, has been proved statistically. It is naturally much better if both operations, intubation and tracheotomy, can be performed in a hospital or a nursing home, for it is only there that the patient can be or is carefully watched.

For the rest we refer—as regards both intubation and tracheotomy—to the chapter on Stenosis. (See p. 417 et seq.)

5. TYPHOID (ENTERIC FEVER).

Etiology and Pathology.—In some epidemics enteric fever has a marked inclination to affect the larynx, where it is apt to produce a series of more or less severe symptoms. This tendency has led some authors to assume that a particular variety of enteric fever, viz., "laryngo-typhoid," exists. The disposition of the larynx to be affected in typhoid fever obviously contrasts with the comparative immunity of the nasal mucous membrane. Setting apart the relatively frequent bleeding from the nose, it might be taken as a rule that typhoid never starts with a coryza.*

The changes affecting the larynx are either specific or non-specific. Owing to the local invasion of the typhoid bacilli, *specific changes* take place at certain foci, which, like the intestinal follicles ("*Peyer's* patches"), contain adenoid tissue. The *non-specific* changes, which, on the whole, occur much oftener, are due to the effect of the toxins circulating in the body, and are most probably caused by staphylococci or streptococci.

The *specific* affections consist of diffuse or circumscribed infiltration of the adenoid tissue, *i. e.*, of the follicles. The infiltrations then soften and break down, forming larger or smaller crater-shaped ulcers, with undermined, thickened edges. These so-called "typhoid ulcers" are found—corresponding to the presence of adenoid tissue—on the epiglottis, ventricular folds, laryngeal aspect of the arytenoid, and on the posterior laryngeal wall.

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* Translating editor's foot-note: Nevertheless, many cases of "cold," or "cold on the chest," or "slight bronchial catarrh," have ultimately turned out to be early cases of enteric fever.—F. W. F. R.

The *non-specific* processes are characterised, in their mildest forms, by a patchy redness, such as occurs in catarrh. Through the invasion of staphylococci or other microbes a superficial necrosis sets in and leads, especially on the epiglottis, to ulcerations which do not show the undermined, thickened edges as do typical enteric ulcers.

Both kinds of ulcers heal, if they have not gone too far, without the formation of scars. But sometimes it happens that they penetrate in depth, from the action of the pus-organisms, and then they cause perichondritic affections of the arytenoid or cricoid cartilages, and in other cases abscesses accompanied by oedema may supervene and result in necrosis, with the formation of cicatrices, and consequently in stenosis.

Occasionally a secondary infection by diphtheria bacilli occurs, leading to the formation of pseudomembranes, and sometimes also to paralysis of the vocal cords.

Symptoms.—In mild cases the subjective symptoms are insignificant; in severe typhoid, however, they are not usually complained of, on account of the coma; otherwise the patient reports pain on swallowing or slight hoarseness, which points to a laryngeal complication. In severe cases, where the laryngeal changes are spread over a large area, the swelling and oedema are very pronounced, or perichondritis or abscess may supervene, and stenosis even, with all its consequences, may result.

The *non-specific* changes usually occur between the sixth and seventeenth day. The *specific* changes in the adenoid tissues occur between the eighth and tenth day, and exactly correspond with similar changes in the intestines. Severe complications, such as perichondritis or abscess, on the other hand, only occur comparatively late in the course of the enteric fever.

Diagnosis.—The typhoid laryngitis is often overlooked because a laryngoscopic examination is often impossible in the cases of highly feverish and comatose patients. In other cases, perhaps oftener than is thought, the symptoms are masked by the apathy of the patient, especially if they are not very marked. The changes, and in particular the ulcers, are, however, so characteristic that their discovery in a patient, feverish from an unknown cause, should always direct our attention to enteric fever.

On the other hand, the typhoid nature of the laryngitis ought not to be mistaken if all the other general symptoms are taken into consideration.

Prognosis.—The prognosis is not altogether unfavourable. Superficial ulcers often, and deeper ulceration nearly always, heal up in the course of the disease or afterwards. But we should always be prepared to see very unpleasant complications, such as sudden oedema, if the ulceration is extensive.

Treatment.—With regard to the patient who is seriously ill, any local treatment should be avoided, and the main thing is to attend to general treatment. Perichondritic abscesses ought to be opened by means of a laryngeal knife. Severe dyspnoea requires tracheotomy. Stenosis caused by contracting scars must be treated according to the lines already indicated.

6. TUBERCULOSIS.

Etiology.—Laryngeal and tracheal tuberculosis do not often occur primarily, and they nearly always follow tuberculosis of the lung in about a third of all the cases of pulmonary disease. The larynx is affected by tuberculosis in various ways, but most frequently by the secretion derived from diseased lungs, which is apt to stick in the pockets and folds of the posterior laryngeal wall, where it is supposed to cause relaxation and erosion of the surface of the mucous membrane, thus preparing the way for the ubiquitous tubercle bacilli. The invasion of tubercle bacilli is, however, not necessarily the result of the erosive action of the sputum.

It has been shown that the male sex suffers more than the female from laryngeal tuberculosis, especially between the twentieth and fortieth years of age, when the larynx is supposed to be most exposed to external harmful agencies, viz., alcohol and tobacco, which are said to set up irritation, which subsequently results in lesions of the mucous membrane. It has further been contended that persons suffering from lung disease are predisposed to catarrhal laryngitis, which is supposed to alter the epithelium in some way or other. These alterations may be so insignificant that they escape notice, and this is probably the reason for the statement that tubercle bacilli are capable of penetrating through the intact epithelium, a view which is in agreement with the recent theory of *Behring* explaining infantile intestinal infection. According to *von Behring,* the so-called primary tuberculosis of the lung in adults is finally to be traced to an invasion of tubercle bacilli, which is supposed to have taken place during infancy; having entered by the intestine, chiefly by the agency of impure milk, the bacilli made their way through the "*large pores*" of the intestinal epithelium, and were thus ultimately transmitted by the lymph-stream into the lung.

However this may be, one may take it as certain that the infection by tubercle bacilli takes place almost always at the posterior laryngeal wall. This harmonises with the fact that this region is the seat of predilection of tubercular changes, and often remains for a long time the only part affected. In some other cases, as, for instance, in miliary tuberculosis of the larynx, the tubercle bacilli are transmitted by way of the blood or lymph-vessels from tubercular glands in the neck or bronchi. Such also would be the result—*e. g.*, infection by way of the lymph-vessels—in those cases of unilateral laryngeal tuberculosis where the lung is likewise unilaterally or is chiefly affected on one side only.* Some authors suggest that pulmonary and laryngeal tuberculosis also is due to aërial affection of the bronchial glands, *i. e.*, by the pulmonary lymphatic glands at the root of the lung.

Pathology.—The tubercle bacillus displays its *specific* action in the subepithelial layer of mucous membrane, where it leads to the formation of "tubercles" of the size of a millet-seed—the so-called miliary tuberculosis, which has been already described in tuberculosis of the nose. The tubercles are embedded in a vascular granulation tissue, which also penetrates into the submucous tissues and between the glands and intrinsic muscles. The glands and vessels undergo various changes, and are displaced, or even destroyed. The tubercles coalesce and form infiltrations which diffusely pervade the tissues or are more defined and circumscribed, forming the tumour-like prominences, or tuberculomata. These undergo retrogressive degeneration, soften, break down, and give rise to a caseous material. The overlying epithelium also softens, becomes fattily degenerated, and is then cast off, and thus smaller or larger sized crateriform ulcers are produced, according to whether only single superficial miliary tubercles or larger conglomerations have been concerned. The single scattered ulcers are also described as "lenticular." These small ulcers may unite and so produce an extensive ulceration. The ulcers show undermined edges, owing to the fact that the submucous tissue breaks down quicker than the mucosa, and the edges of the ulcers are notched or serrated, because they are the seat of fresh tubercles, which in their turn sooner or later also break down. The formation of granulation tissue is common to all tubercular ulcerations, on their floor, as well as on the edges of the ulcer. One may, therefore, see the margin of tubercular ulcers occupied by either small yellowish tubercles or red granulations, or both together. The red granulations not infrequently grow so abundantly, that they form actual papillary excrescences which have the appearance of being papillomata.

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* We wish to emphatically point out that in left-sided affection of the lung the right side of the larynx might be affected, and vice versâ, and that in unilateral tuberculosis of the lung both halves of the larynx may be diseased; nevertheless, it is possible to observe, in a great majority of cases, that the respective affections show certain uniformity with regard to the side affected.

If the process advances, it also penetrates in depth, and may attack the perichondrium, causing perichondritis, which denudes the cartilage, and since this is consequently bereft of its nourishment, it then becomes necrotic. This process is usually accompanied by oedematous swelling of the adjacent tissues. Perichondritis and chondritis must be laid to the account of the pyogenic cocci, which cause secondary infection and act either alone or in company with the tubercle bacilli. The severer forms of tuberculosis are nearly always caused by such a mixed infection.

If the intermuscular and intramuscular tissues become infiltrated, the muscular substance itself degenerates, which causes disturbances of the voice, but even the nerves themselves may also become affected.

In the trachea, tuberculous infiltration, owing to the firmer adherence of the mucous membrane to the trachea, is not as conspicuous as it is in the larynx. Ulcers may be present in small number, but of large size; or they are multiple and then mostly small, and are widely distributed over the surface. The trachea is usually not seriously altered, unless the laryngeal and pulmonary disease is far advanced, and it is for this reason that the pathological changes produced by tuberculosis have been studied rather on the post-mortem table than intra vitam. Per se, they hardly exercise any marked influence upon the course of the disease; which is, however, defined by the laryngeal and pulmonary affection.

The chief *non-specific* alteration, apart from the perichondritis and chondritis caused by a secondary (mixed) infection, are considered to be the conspicuous anaemia of the mucous membrane and the chronic laryngeal catarrh. Both evidently in my opinion are the effect of a dyscrasia due to the toxins. Possibly the catarrh has already been in existence in many cases, and has fostered the specific infection (see above). Anyhow, it is certain that chronic laryngitis is a regular accompaniment of laryngeal tuberculosis, and frequently also of pulmonary tuberculous disease. (See pp. 454 and 477.) Laryngitis is very often the only symptom in the course of "consumption" pointing to an infection of the larynx, and is usually distinguished by its obstinacy.

Histological Changes.—The epithelium over an infiltration is very little altered; but sometimes it may be found to be thickened and warty, especially if the infiltration is close under the epithelial layer. In such a case single or multiple round-celled accumulations may be seen immediately below the epithelium. These are the well-known "tubercles," which usually contain one or several giant-cells among the round-cells.

The "tubercles" are themselves situated in an area of small round-cells, which surround the vessels and glands. An ulcer, of course, shows loss of epithelium, and the small-celled infiltrations accordingly look as if they were "dug out" or puckered. Bacilli are not always discoverable in quantities, and they are the less numerous in proportion to the greater number of giant-cells seen.

Symptoms and Course.—Laryngeal tuberculosis begins with a series of symptoms which, neither subjectively nor objectively, offer anything characteristic, and are often masked by the coexisting pulmonary disease. Patients now and then complain of some hoarseness or only of slight roughness of the voice, "hardly worth speaking of," or that the voice soon becomes fatigued. They experience various sensations in the larynx, such as a feeling of pressure as of a foreign body in the neck, of soreness, burning, or tickling, irritation, etc. To account for all these manifold sensations, nothing but a marked pallor of the mucous membrane can be discovered laryngoscopically, though this may often be already obvious in the pharynx, and is often, though not always, accompanied by slight symptoms of pharyngeal catarrh. In my opinion, as already pointed out, the pallor is the first manifestation of the effect of a disturbance of nutrition due to the toxins, but yet not of a decidedly specific nature. In due course the symptoms gain in intensity and become more distinct.

(*A*) *Subjective Symptoms.*—They are naturally dependent on the seat and extent of the tuberculous process and on the lesions thereby produced. The voice, which at first was perhaps only impure, becomes rough, hoarse, or even aphonic. This is due to the pathological changes on the vocal cords, ventricular folds, or arytenoid cartilages and posterior wall of the larynx, which impede the normal vibration or adduction of the vocal cords, though the muscles themselves might be degenerated or the recurrent nerve be paralysed as the result of pressure from swollen bronchial glands or tuberculous processes in the apex of the lung. At first it is usually due to paresis and weakness of the muscles on account of the pallor of the mucous membrane by which the voice is altered, and the cough is mainly caused by the coexisting lung disease. Next after this alteration of the voice, swallowing becomes painful (dysphagia). This arises from diffuse infiltrations on the epiglottis, but still oftener from ulcers on the epiglottis, arytenoids, and posterior laryngeal wall, or even from perichondritis. It may assume such a degree that feeding may become exceedingly painful and difficult, so that only a certain amount of fluid food can be painfully swallowed. Moreover, misswallowing is very common, or the poor patients, with painful coughing and retching, vomit up all that they have partaken of.

In unilateral disease the pain radiates towards the ear of the same side, and in bilateral affection into both ears. In advanced cases every single cough and every sound made is painful. The whole state of subjective discomfort is much aggravated if dyspnoea ensues. Partially caused by the lung affection, the dyspnoea becomes very much worse if the larynx, on account of oedema and infiltration, becomes narrowed. As has been said in the general part of this section, the stenosis developes gradually, so that the patients may become used to the restriction of their oxygen supply. On the other hand, also, perichondritis and oedema are apt to give rise to sudden stenosis. Expectoration is often very painful and impeded by the oedema.

Thus all the symptoms finally form a picture which, taken together with the conditions exhibited by the lung, will leave no doubt of the nature of the disease. In accordance with the subjective symptoms, so are the objective, as is shown by the laryngoscope.

(*B*) *Laryngoscopic Appearances.*—Four forms of disease may be discerned, all corresponding to the pathological changes: (1) *Tuberculous infiltration.* (2) *Tuberculous ulcer.* (3) *Tuberculoma.* (4) *Miliary tuberculosis.* To these forms we may add a fifth: *tuberculous perichondritis and chondritis.* The first two named are exceedingly common, but the latter forms are seldom observed. In many cases these various forms combine, especially in advanced cases, and provide a greatly varied laryngoscopic picture. The posterior wall of the larynx is the most common seat of tuberculous disease; next to it come the vocal cords, and the least often affected is the subglottic region; in between these come, in about equal proportions, the ventricular folds, the arytenoid cartilages, aryepiglottic folds, and the epiglottis.

1. *Tuberculous Infiltration.* (See Figs. 163 to 166.)—As has been said, the posterior wall, viz., the interarytenoid region, is the seat of predilection, and it is oftener subject to tuberculous infiltration than all the rest of the larynx put together. It is, therefore, safe to say that *infiltrations, and therefore ulcerations, of the posterior wall are almost pathognomonic of tuberculosis.*

The infiltration is usually of an opaque greyish colour, and is situated in the middle line or a little towards one side. It is usually broad, more or less prominent, flattened or conical, or sometimes even tuberous or serrated. In the latter case it looks almost like a papilloma, behind which an ulcer might be hidden. But diffuse infiltration is sometimes nothing else than the broad tumefied margin of an ulcer situated behind or below it, and which may be made visible by *Killian's* method of examination. In some cases, however, an ulcer may be suspected with certainty if pus is discovered behind the infiltration. In all these cases one should always examine by *Killian's* method, if for no other reason than to form a correct opinion concerning the dimensions of the infiltration. One will often be surprised to see how much the infiltration really exceeds the first estimate.

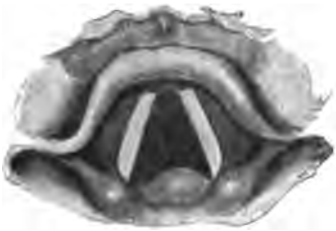

Fig. 163.—Broad-based infiltration of the posterior laryngeal wall.

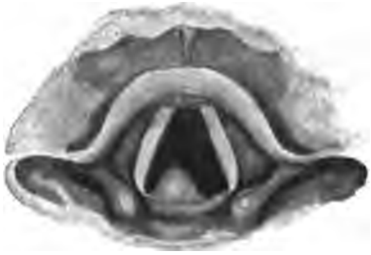

Fig. 164.—Conical infiltration of posterior wall.

Sometimes the infiltration is so large that it prevents adduction of the vocal cords, or gives rise to dyspnoea, and at other times again it might be so insignificant that one may have doubts as to whether it is not a simple thickening due to catarrh. (See p. 451.)

Infiltration of the vocal cords and ventricular folds at the beginning is almost always *unilateral*, and appears as a diffuse pink swelling, rendering the vocal cord swollen and thick, while the ventricular fold projects like a pad into the lumen covering totally or partially masking the vocal cord beneath it. In some cases only a part of the vocal cord is infiltrated, and then mostly the region of the vocal process.

The presence of such a unilateral redness and swelling points, as a rule, to tuberculosis, and is against its being due to simple catarrh, which is almost always bilateral.

The *arytenoid cartilages* and *aryepiglottic folds* also are infiltrated on one side only, but not infrequently on both sides, as is also the case with the *epiglottis*.

The infiltrated parts are much disfigured. If the entire epiglottis is diseased, it looks like a turban. If the region of the arytenoid cartilage is affected, it looks blown out, like a bladder. The aryepiglottic fold becomes a sausage-shaped pad. The configuration of all these parts is still more disturbed by oedema of the adjacent parts, which, in addition, very much hinders the movements of the vocal cords. Subglottic infiltration is rare, but, on the whole, it shows the same picture as subglottic laryngitis due to catarrh.

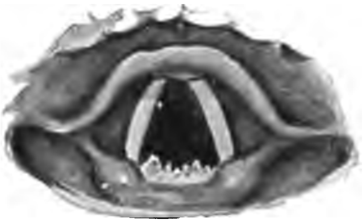

Fig. 165.—Serrated infiltration and papillary excrescences of the posterior wall.

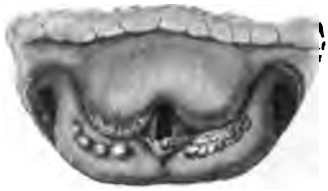

Fig. 166.—Infiltration of the epiglottis, aryepiglottic folds, and posterior wall. The infiltration of the right aryepiglottic fold shows tuberous prominences. On various places ulceration has taken place (*Türck*).

2. *Tuberculous Ulcer.* (See Figs. 167, 168.)—The form and size of the ulcers are very varied, and depend essentially on the infiltration, from whence they are derived by a process of caseous degeneration. Not infrequently parts of the old original infiltration may be seen, especially in cases of deep ulceration, where it forms part of the everted margins of the ulcers. On the posterior laryngeal wall the ulcer may be even concealed by such an infiltration (see above). In many cases, on laryngoscopy, by *Killian's* method of examination, one sees at first only the excrescences already mentioned, and it is found that these excrescences arise from the floor or edge of a large ulcer.

Ulceration spreads from the posterior wall in all directions, but mostly on to the arytenoid cartilage, where it expands, especially on to the inner surface of the vocal processes and on to the vocal cords.

One sees, at first, small flat single or multiple ulcers, situated on the surface or margin of the vocal cord, and in such a case the vocal cord appears notched, serrated, "gnawed."

The elastic fibers of the vocal cords resist for a long time the ulcerative process. Consequently, we see the superficial ulcers often forming longitudinal, gutter-shaped grooves,—the so-called "lip ulcers,"—by which the vocal cord appears divided into two or more sections. The marginal ulcers penetrate in depth, destroying considerably the vocal cord, of which ultimately they do not leave much behind.

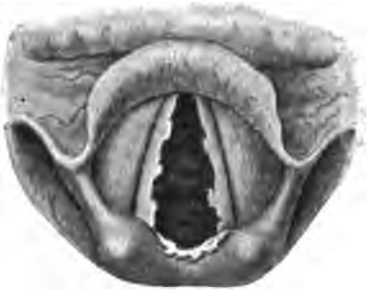

Fig. 167.—Tuberculous ulcers on both vocal cords and posterior wall. The vocal cords show a serrated edge.

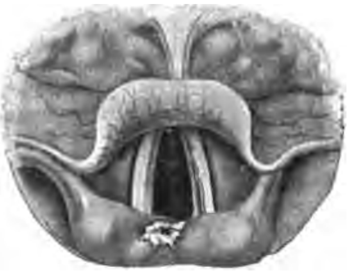

Fig. 168.—The left vocal cord appears divided into two parts by a notch-shaped ulcer (lip ulcer). The posterior wall, which is swollen to a great extent up to the arytenoids, shows an irregular, notched ulcer.

The tuberculous ulcer of the *vocal process* is frequently seated on its inner surface, and, owing to the thin covering of the cartilage, soon leads to perichondritis (see below). At the beginning it is difficult to recognise whether the cartilage is already involved or not, and laryngoscopically, the perichondritis itself, in its first stage, is hardly distinguishable from an infiltration over the arytenoid cartilage. Like the arytenoid cartilage, the cricoid plate may also be the seat of ulceration, which has spread from the posterior wall (perichondritis cricoidea). If such is the case, the posterior wall is enormously swollen, as is also the subglottic region, causing dangerous dyspnoea.

On the ventricular folds several flat ulcers may be noticed, which, in the later stages, unite and form a large, irregular ulcer, which is likely to invade the adjacent tissue.

On the epiglottis one usually sees marginal ulcers; those on the laryngeal surface often escape discovery on account of the infiltration and rigidity of the structure. In many cases a perichondritis of the epiglottis is the cause of the swelling. The ulceration in the subglottic region, and in the trachea likewise, very often escapes discovery.

3. *Tuberculoma.* (See Figs. 169 and 170.)—The tuberculous tumour of the larynx sometimes occurs on the posterior wall and ventricular folds, forming a round, smooth tumour, looking like an infiltration, with the difference that the infiltration merges gradually into the adjacent tissue, while the tuberculoma is more or less distinct from its surroundings, like a new-growth.

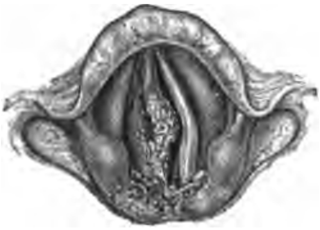

Fig. 169.—Tumour-like ulcer of the right vocal cord, covered with granulations. Large ulcer on the posterior wall (*Türck*).

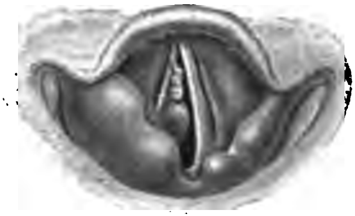

Fig. 170.—The right vocal cord is irregularly infiltrated, giving the appearance of a tumour, with ulceration. Oedema of the right aryepiglottic fold. The larynx is stenosed (*Türck*).

4. *Miliary Tuberculosis.*—Miliary tubercles may be met with, and they look like the small tubercles so often seen at the edges of a tubercular ulcer. Miliary tubercles are, however, seldom seen, probably for the reason that the minute nodules rapidly break down.

5. *Tuberculous Perichondritis and Chondritis.*—The implication of the perichondrium, and of the cartilage itself, is regarded as the worst complication of tuberculous laryngeal disease, and

rightly so. (See p. 458.) It not only causes violent pain on pressure from outside and on swallowing, and disturbances of the voice, but the pronounced swelling, in combination with the oedema of the neighbourhood, is capable of giving rise to the most dangerous dyspnoea. Most commonly the arytenoid cartilages are affected; next comes the cricoid and epiglottis; and least often affected is the thyroid cartilage.

On examination with the mirror one finds a shiny, oedematous swelling, which often extends beyond the focus of the disease, and which cannot at first be differentiated from an ordinary infiltration. The ulcer, which has caused the perichondritis by its spreading, very often escapes observation, as do also the vocal cords, which may be partially or totally covered and obscured. If one is able to see them, one will at once notice the difficulties of their movements, which may even increase so as to amount to complete immobility. The diagnosis will become assured if pus can be detected and the denuded cartilage becomes visible. The coughing up of a portion of the necrosed and cast-off cartilage will be a certain proof.

(*C*) *The course of the disease* will naturally be different, according to whichever part happens to be the seat of the disease. Generally speaking, *the laryngeal process runs parallel and equal with the pulmonary disease.* Not infrequently the laryngeal affection plays quite a subordinate rôle, and in the majority of cases it is the pulmonary tuberculosis which settles the fate of the patient. In some cases, however, the laryngeal disease may in the long run prove the decisive factor, viz., if the increasing dysphagia limits more and more the possibility of feeding, or if the dyspnoea assumes such a degree as to threaten suffocation. We do not deny that sometimes the laryngeal tuberculosis, without any recognisable cause, or under the influence of the medical treatment, shows a halt in its advance. Usually, however, this pause is followed by a rapid recrudescence. Pregnancy, in particular, according to the observations of *Kuttner*, *Kaminer*, and *Baginsky*, exercises a very malign influence upon laryngeal tuberculosis, and this may be regarded as an indication for the artificial termination of the pregnancy if not too far advanced. *Sokolowsky* goes so far as to advise the examination of every female patient who may be suffering from laryngeal tuberculosis for signs of pregnancy, so as not to miss the opportunity for inducing premature labour.

Diagnosis.—If the signs in the larynx are distinctly marked, and if disease of the lung is known to exist, then the diagnosis offers no difficulty. But diagnosis at the commencement of the disease is not easy if the signs in the larynx are doubtful and the examination of the lung proves negative. The laryngeal changes may be so insignificant that they do not lend themselves to the diagnosis of tuberculosis. This may be especially said with regard to the anaemia, which is admittedly very often found in tuberculosis. But it is striking how many apparently healthy individuals, and again how many persons with non-tuberculous laryngeal affections, show the same anaemic condition of the laryngeal mucous membrane. Anyhow, any noticeable anaemia of the pharynx and larynx, especially if associated with certain subjective symptoms, such as hoarseness and paraesthesia, must always render us suspicious of tuberculosis; and if it has not been already done, calls for a very careful examination of the lung. These suspicions would be increased if, besides the anaemia, symptoms of inflammation on one vocal cord only, or on one side of the larynx, were found, or if a swelling occurred on the posterior laryngeal wall.

Signs of unilateral inflammation should always direct our thoughts to tuberculosis, if they cannot be traced to traumatic causes. What may at first appear to be only a simple catarrh of the vocal cord may very often be the first sign of the beginning of an infiltration. It is, however, true that commencing tuberculous infiltrations are difficult to recognise. The infiltrations of the posterior laryngeal wall, which are rightly considered as characteristic and typical of laryngeal tuberculosis, are at first so slightly developed that they may be easily mistaken for the simple thickening of a catarrh. (See p. 451.) Their opaque greyish colour is deceptive, as catarrhal pachydermia may show just the same opaque grey appearance; and, on the other hand, infiltrations may also be red. It even happens that the infiltrations may be covered by thickened epithelium, if, for instance, the infiltration lies close up to the surface. (See p. 482.)

Matters are usually so shaped that, besides these little characteristic symptoms, one is able to see something more definite—for instance, an ulcer on a vocal cord. Small, superficial ulcers or erosions may also be found in catarrh, but a *distinct or perhaps deep ulcer always points to tuberculosis or syphilis.*

If pulmonary lesions have been discovered, the ulcer in the larynx may be regarded as being tuberculous; otherwise the discrimination between a syphilitic and a tuberculous ulcer is not easy. As regards the differential diagnosis many points have been emphasised in respect to the site, extent, and course of the disease. Thus it was pointed out that tuberculosis decidedly affects the posterior laryngeal wall, while syphilis mostly occurs in the aditus laryngis. Tuberculous infiltrations usually break down later than syphilitic ones. In syphilis the mucous membrane, especially around the ulcers, is generally reddened, while in tuberculosis it is pale and anaemic. Lastly, syphilitic ulcers penetrate more in depth, are sharply cut (punched out), and are covered with a smeary film, whereas tuberculous ulcers are flat, spread out more superficially, irregular, and covered with granulations. Pain also may be quoted, viz., tuberculous ulcers are very painful, but syphilitic ulcers are conspicuously indolent. Thus one could construct the following table:

Tuberculosis.	Syphilis.
Site: Mostly on the posterior wall, vocal cords. and arytenoid cartilage; seldom on epiglottis.	Mostly on epiglottis.
Mucous membrane: Usually pale.	Often bright red.
Ulcer: Spread out superficially; edges irregular, notchy; granulations; miliary tubercles; painful.	Penetrates in depth; edges sharp (punched out); floor is smeary; painless.
Infiltration: Slowly developing.	Rapidly breaking down.
Body: Pulmonary diseases, cough, etc.	Other signs of syphilis in pharynx, mouth; rash, etc.
Microbes: Tubercle bacilli.	Spirochaeta pallida.

All the foregoing landmarks, if combined, would be sufficient in any given case; but frequently they are so little pronounced that one cannot rely on them. The same may be said of the pain; syphilis is, without doubt, a very painless disease, while tuberculous ulcers are rather tender. Here again personal sensitiveness plays a great part, and, apart from this circumstance, the pain itself depends much on the seat of the ulcer. If, by examination of the whole organism, no decision can be arrived at, we may try to make a microscopic examination of the sputum or of the secretion removed by means of a curette. Whether the finding of the spirochaeta pallida is a proof of syphilis we leave others to decide. In any case, only a positive result is a proof. Histological investigation might be useful in many, but not in all, cases, and there remains only the probationary trial of an antisyphilitic treatment, if one does not feel inclined to resort to the use of "tuberculin." It should, however, be borne in mind that mixed infection of tuberculosis and syphilis may occur, and that occasionally *both might be complicated by carcinoma.* In the latter case a histological examination of a portion of the tumour, excised for this purpose if needs be, must be resorted to and repeated more than once. Occasionally leprous infiltrations or diabetic ulcerations (diabetic furunculosis laryngis) occur, which must be recognised. Ulcers due to caustics or scalding also have been observed, and have been mistaken for tuberculous ulcers.

(For the differential diagnosis between tuberculosis and lupus see p. 500.)

Prognosis.—One should never give up, says *M. Schmidt*, a phthisical patient as long as he has a good digestion and a strong heart. This then is also the essential point in laryngeal tuberculosis, viz., the general condition of the patient, and, of course, the condition of his lungs. But the laryngeal disease, inasmuch as it may not have already gone too far, sometimes shows, independently of the lung disease, a distinct improvement, and the morbid changes may recover to such an extent that they might be spoken of as cured. This, however, is only a local cure, and we desire to emphasise that it is often only a cure from the clinical point of view. We do not deny that a tuberculous ulcer may be cured. I myself remember a forester, in whose larynx a large tuberculous ulcer was found on the posterior wall, spreading on to the vocal cord and arytenoid cartilage. This ulcer completely healed under partial curettement and cauterisation with lactic acid; but this case I consider a great exception to the general run. Sooner or later a relapse occurs, and the more so if the condition of the lungs does not improve. It is, however, worth noting how great, in some cases, is the resisting power of the patients, even in far-advanced laryngeal tuberculosis, and for what a long time they can continue to battle with the dyspnoea and stenosis. Taken altogether, the prognosis of the functional disorders caused by the tuberculous process is favourable; and, as regards the case as a whole, it is very doubtful, indeed, even in the most favourable.

Treatment.—In the treatment of laryngeal tuberculosis three points have to be considered: (1) *The general condition of the patient;* (2) *the lungs;* (3) *the changes in the larynx.* Just as one or the other item predominates, so the treatment must be directed on general lines with due consideration for the individual propensities, or they may be combined with certain local measures.

(*a*) *The general treatment* is contained in that for tuberculosis of the lung, and aims, in modern conception, at putting the patients under the most favourable hygienic and dietetic conditions possible. With regard to the *health resorts*, if the patient is able to travel, the laryngeal affection is not the most important thing. It is essential for the patient to be in a pure, dust-free atmosphere; one patient does better at the sea, another, in the mountains. Here, as always, individual predisposition as well as constitution are the important factors. Where the financial position permits, the patient can be sent during the winter to a place of an equable and mild climate, such as the Riviera, Ajaccio, Madeira, Algiers, etc. To stay in a good sanatorium suitably situated is still better, where a silence-cure can be strictly enforced (*Felix Semon; Lublinsky*). According to *Semon*, a silence-cure, under certain circumstances, combined with appropriate local treatment, is suitable in cases of inflammatory irritation of the larynx from tuberculosis, in obstinate catarrh, congestion of the vocal cords, infiltration of the ventricular folds, for circumscribed ulceration of the vocal cords, or the interarytenoid region, and lastly in diffuse infiltration and disorders of the crico-arytenoid joint.

Besides resting the larynx, every other form of irritation, as has been shown in the General Section of this Part (p. 391, et seq.), should be avoided, and various drugs, if necessary, must be applied. In any case, the center of gravitation in treatment lies in strengthening the patient, and, according to individual taste, fixing an appropriate diet. *Qui bene nutrit bene curat.*

(*b*) *Local treatment* is carried out according to the stage at which the laryngeal and pulmonary disease has arrived.

1. If the process in one or both organs is already far advanced, and the patient in a bad and hopeless state, then local treatment, with few exceptions, should be omitted, for the patient is not helped by it, but may have his only small remaining powers of resistance thereby diminished. That being so, we must proceed symptomatically and apply narcotics. Pain on swallowing, which may exist, or dyspnoea, may be mitigated by instillation of menthol oil, ½ to 1 per cent., by syringe; or, if the patient feels strong enough, inhalations of menthol vapor by special vaporisers (see p. 398), may be administered. Menthol oil is first instilled as a 20 per cent. oily solution; later on, the concentration may be increased to 50 per cent. In the intervals between the instillations the patient should take menthol pastilles, or so-called angina pastilles (5 or 6 a day). Shortly after the instillation, the patients should take some milk food, for fluids are apt to irritate. In some cases one can do no good except by spraying a 10 to 20 per cent. solution of cocaine before each meal, having previously instilled a few drops of adrenalin or insufflated renoform powder. If the pain is caused by large ulcers, then insufflations of orthoform are beneficial, but if the mucous membrane is intact, this remedy fails. The toxic by-effects which, nolens volens, may be produced by the continued administration of the foregoing drugs, do not appreciably fall into our consideration, in the face of the desperate straits of the patient. In threatening dyspnoea, tracheotomy is required.

2. If the general condition of the patient is *satisfactory*, and should neither *cachexia* nor *fever* be present, or if the disease in the lung shows no inclination to rapidly progress; and, lastly, if we find in the larynx *well-defined* alterations or such as are *amenable* to therapeutic measures, then it may be allowable

to undertake a line of local treatment which may essentially consist in removing the tuberculous focus by surgical means; or, in rendering it innocuous, by means of caustics. As may be understood, neither the one nor the other method is sufficient to insure success. I say, "it may be understood," for it is very often difficult, nay, impossible, to estimate, in any given case, by the laryngoscopic image, how far a tuberculous ulcer or infiltration has extended, and what will be the reaction of the laryngeal tissue to the operation or manipulation performed. It is true that just this reaction, *i. e.*, the natural recuperative power, varies greatly in different cases, as it might be very feeble in cases which, in our opinion, may show a favourable course; and, on the other hand, there are cases which are obviously bad, yet show a remarkable tendency to recover from the effects of an operation. To judge from all that has been said, it will not be easy, in many cases, to arrive at a precise conception of what should be done with regard to local treatment. The possibility of arresting tuberculous processes in the larynx for a short or long period must be admitted; nevertheless, the value of local treatment should not be overrated. It is also certain that superficial and not too far-advanced ulcers heal spontaneously, if only the patients take care of themselves and their larynges, and if they are properly fed and nourished, move in pure air, and, above all, *keep absolutely silent!** If, in such a case, the application of a drug has appeared to have helped in the recovery, who is able to prove it? Anyway, the fact that under favourable conditions a tuberculous ulcer may heal spontaneously, reminds us that we should not be too liberal with the administration of medicamental salvation; and that we should not apply caustics too early or too freely.

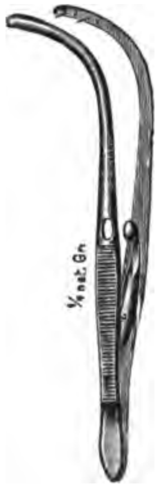

Fig. 171.—Pressure forceps (after *Krause*).

Innumerable remedies have from time to time been recommended, a sign of how doubtful their effect may prove to be. Of all these, it is perhaps *lactic acid* which has found the best and widest acceptation. According to *Mosetig*, it destroys the fungating (granulation) tissue only, while it leaves the healthy mucous membrane intact. One is accustomed to apply it for deeply spreading ulceration or in those superficial ulcers which show no tendency to heal, in spite of appropriate general treatment. The concentration is made to vary from 20 to 80 per cent., according to the sensitiveness of the patient; and it is applied by means of a sponge-holder or *Krause's* pressure forceps (see Fig. 171), the laryngeal mucous membrane having been previously anaesthetised. After the cauterisation, it is then only necessary to look for the white eschar; and the proceeding can be repeated after a few days, when this has been cast off.

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*Translating editor: "Rest in pain" (Hilton) being as useful for a diseased larynx as for a broken leg.—F. W. F. R.

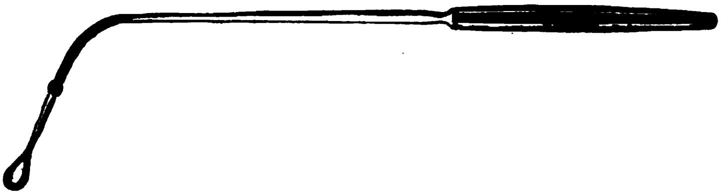

Fig. 172.—Curette with revolving end for the posterior laryngeal wall.

Infiltrations sometimes subside by themselves; this fact, however, should excite our doubts in many a case as to whether we really had to deal, after all, with a tuberculous infiltration. In this disease, treatment by drugs is usually a failure, and in many cases it will only serve to accelerate the breaking up of the infiltration and thus open up fresh pathways for bacteria, especially for streptococcal and staphylococcal invasion, which exercises so bad an influence on the disease.

We must now turn to *the surgical treatment of laryngeal tuberculosis*, which is *eminently intralaryngeal*, and only exceptionally *extralaryngeal.*

Intralaryngeal surgery comprises curettement, galvano-cauterisation, and electrolysis.

(*a*) *Curettement*, the technique of which is not so easy, is performed with scraping or cutting instruments, called *curettes.*

For the posterior laryngeal wall, a simple curette (see Fig. 172) is good enough; for the epiglottis and lateral regions, which are less easily accessible to the scraping instruments, a double

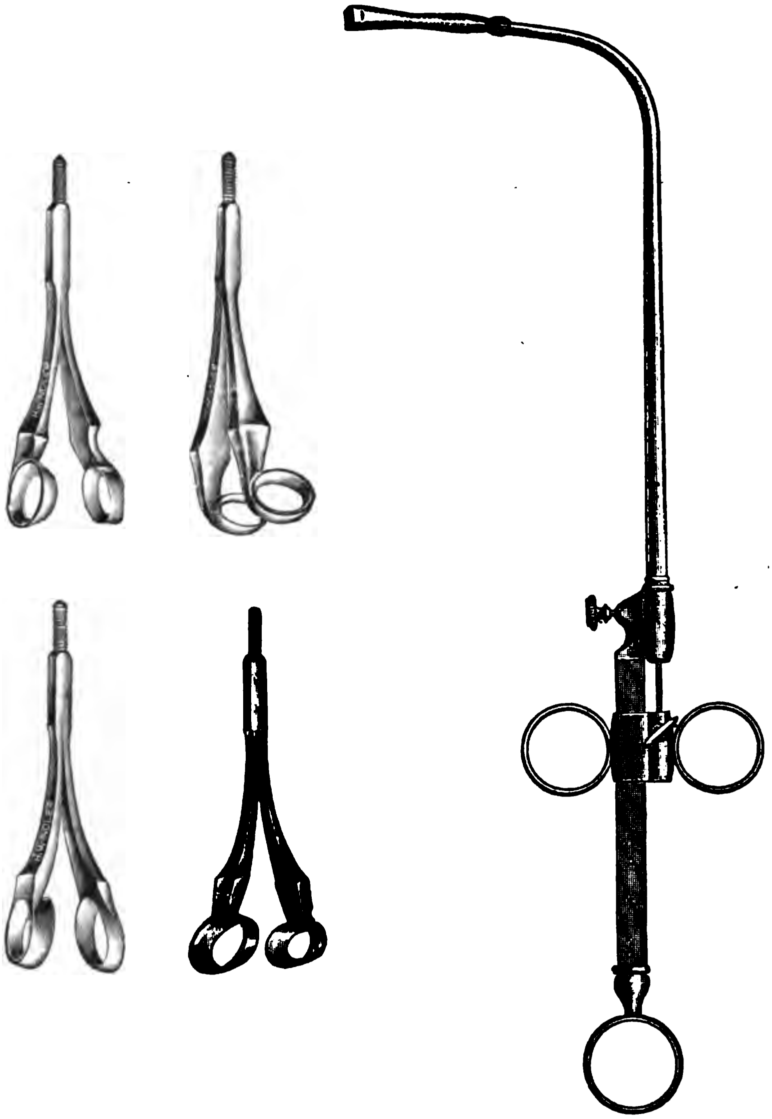

Fig. 173.—Revolving double curettes (after *Krause-Heryng*), with guide tube and wire, fixed on a *Krause's* universal handle.

curette must be used, viz., *Krause-Heryng's revolving double curette* (see Fig. 173). For the ventricular fold, that of *Landgraf-Krause* (Fig. 158, p. 457), or *Rosenberg's* small scooped forceps (p. 458, Fig. 159), is useful. *The curette should be used only* where the ulcer is defined and does not penetrate in depth. It is very suitable for the removal of granulations and excrescences. Infiltrations should be let alone if they are not well *circumscribed* or appear more like a tuberculoma. *The curette is contraindicated* in diffuse infiltrations, in large ulcerations, and in perichondritis. Having performed curettement, the curetted region should be treated with lactic acid, *the epiglottis excepted.* The operation may have to be repeated after eight to twelve days if all signs of reaction have subsided. Pain after operation, usually not severe, may be relieved by ice, menthol, orthoform, etc. Troublesome bleeding is sometimes experienced in operations on the ventricular folds.

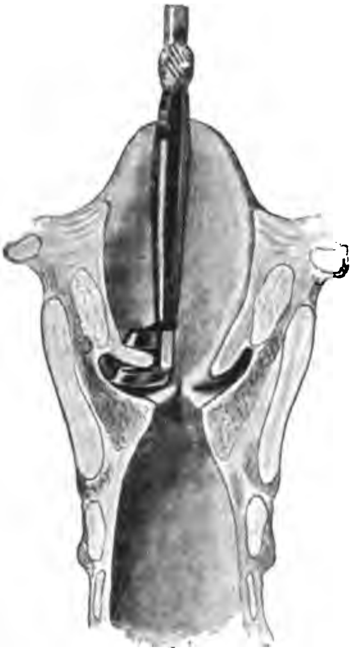

Fig. 174.—Curettement of the right ventricular fold. *Landgraf's* double curette is shown in situ.

In ulceration or infiltration of the epiglottis curettement is indicated under any circumstance, if pain on swallowing is distressing and feeding becomes painful. In such a case removal of the infiltration or ulceration by means of a double curette or sharp scoop-forceps (see Fig. 175) must be done even in the case of patients who are severely ill.

If one does not expect too much from curetting, but carefully selects the patients suitable for the operation, and if one does not in overconfidence therein neglect general hygienic and dietetic measures, then in some cases remarkable results, though not lasting, will be obtained. One must never be surprised by subsequent unexpected and rapid aggravation of the patient's condition.

(*b*) *Galvano-cauterisation.—It is contraindicated in stenosis* of greater degree, on account of the marked reactionary swelling following the operation, and may be left out of account altogether. *Grünwald* has recently revived it in the form of "deep puncture" (galvano-caustic puncture).

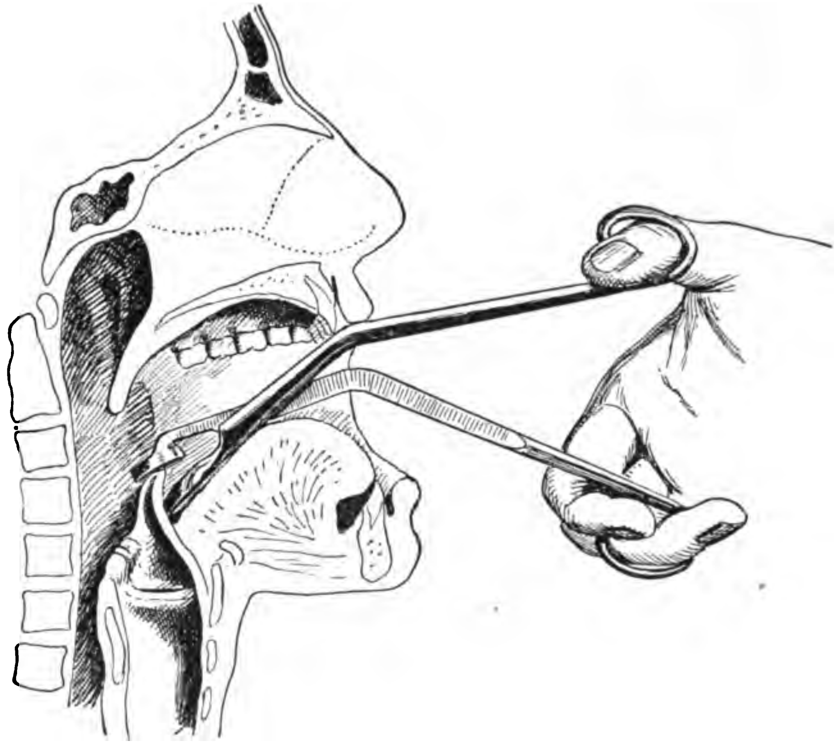

Fig. 175.—Removal of the epiglottis. *Nikitin's* sharp forceps shown in situ (diagram).

(*c*) *Electrolysis* has been used in order to destroy indurated infiltrations, but nowadays it is seldom applied on account of its complicated method and really worse than slow action.

How much tubercular processes in the larynx will be benefited by the *Röntgen* rays in the future—the technical skill and suitable anatomical conditions being granted—cannot yet be stated. *Mader* has recently devised a pharyngo-laryngeal tube which permits the irradiation of the laryngeal part of the pharynx (hypopharynx), and partly also of the interior of the larynx. Sunlight as the actinic source is employed in the method of *Sorgo-Kunwald.*

It presupposes that the patient is able to use the laryngoscope on himself. Seated with the back towards the window, the patient reflects the sunlight into his mouth with the aid of an ordinary toilet mirror; then, in the same manner as is done by a physician, he inserts a laryngeal mirror, and manipulates it until he can see the image of his own larynx in the looking-glass held or fixed in front of his mouth. It remains for the future, however, to decide whether this method of "autoradiation," which depends much on the weather and the season of the year, and which is, moreover, contraindicated in irritative conditions, will find general adoption.

(*d*) *Extralaryngeal Operations.—Division of the larynx* (laryngofissure), followed by scraping away the diseased tissue, has not often been performed. The operation, however, has been warmly recommended. *Total extirpation is inadmissible.* Only in exceptional cases, perhaps where the disease is sharply localised to one side only, could a *partial resection* be performed. *Tracheotomy*—not that which must be performed in any case of threatening dyspnoea, but *early tracheotomy*—is recommended by *M. Schmidt,* in order to afford rest to the larynx and to check the otherwise unceasingly advancing processes. He states that he has seen remarkable improvement following the operation, which, however, is permissible in cases where the lungs are only very slightly affected. Other authorities reject the operation, maintaining that the act of expectoration is rendered more difficult by tracheotomy and accordingly aggravates the pathological process.

APPENDIX.

LUPUS.

Etiology and Pathology.—Lupus, though etiologically belonging to "tuberculosis," produces a series of symptoms and changes in the larynx which are quite different from those caused by ordinary tuberculosis. Histologically, one finds in lupus a vascular granulation tissue containing tubercles with giant-cells, *but tubercle bacilli are very scanty*, a fact which perhaps serves to explain the very slow progress and the obvious tendency to contraction in lupus, as opposed to "tuberculosis."

Lupus is, on the whole, a rare occurrence in the larynx, and arises in younger patients and especially in females. *Chiari* and *Riehl* found, among 70 cases of lupus, that the larynx was affected six times. Usually it is associated with lupus of the nose, throat, mouth, and skin.

Symptoms and Course.—The subjective symptoms are usually insignificant, and contrast much with the objective changes. The patient may only complain of a little hoarseness or tension in the throat, but later, when the process becomes more advanced, dyspnoea may be added.

The process mostly occurs in the aditus laryngis, and especially on the epiglottis. Only in the advanced stages do the lower regions of the larynx become affected. The affected parts are infiltrated, swollen, and slightly reddened, and show on the surface small pale nodules, frequently in clusters, which give the mucous membrane a somewhat granulated (chagrinned) appearance. Later on, fresh crops of eruptions follow, forming papillary prominences, seated mostly on the epiglottis. In other cases the nodules do not project beyond the level of the mucous membrane. One or other of the nodules may become absorbed and disappear; others again soften and break down, forming small or larger sized ulcers, which—except on the epiglottis—do not often show any liability to penetrate in depth, but a distinct tendency to heal up, with the formation of scar tissue. This tendency of forming ulcers and a simultaneous proclivity to cicatrisation is characteristic of lupus. (See Fig. 176.) In the scar or in

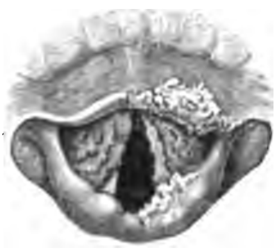

Fig. 176.—Lupus of the larynx (*Chiari*).

Formation of lupus nodules on the left half of the epiglottis and left arytenoid. Both the ventricular folds are occupied by nodules and also the left vocal cord. Of the right vocal cord, only the posterior extremity is visible.

the adjacent tissue fresh nodules, however, soon again spring up.

Laryngoscopically, one finds that the nodules are arranged singly or in groups, and it looks almost like a granulating surface. Later on, circular or irregular ulcers may be seen, as well as scars and papillary excrescences.

Diagnosis.—If one finds nodules, especially on the epiglottis, and also ulcers, together with the formation of scars simultaneously, and these are associated with lupus of the face or throat, then the diagnosis need not be difficult. In other cases the disease will be recognised only after prolonged observation. The differential diagnosis must be made from tuberculosis and syphilis, and in certain cases from carcinoma. Tuberculosis, if accompanied by ulceration, causes much more trouble, and never shows the same tendency to cicatrise as is so characteristic of lupus; it almost always occurs in conjunction with pulmonary tuberculosis, whereas lupus only occasionally does so. Syphilis, it is true, occurs mostly on the epiglottis, and is also prone to contraction, but in this case, however, an antisyphilitic treatment will soon unveil the mystery. A confusion of "tuberculosis" with "lupus," however, would be no great calamity, considering the near relationship of both diseases.

Prognosis.—Although the course of lupus is of a very slow progress and it preserves its local character for a long time, the prognosis is, nevertheless, unfavourable. One can never tell to what extent the tissue may be destroyed and whether the lung may not be affected. Apart from the outbreak of a complication on the part of the lung, stenosis from excessive growth or cicatricial contraction must sooner or later be expected.

Treatment.—Ulcers may be cauterised with lactic acid (20 to 80 per cent.) or tincture of iodine. Prominent papillary excrescences, if accessible, may be removed by the curette, and afterwards rubbed with the drugs before mentioned. Internally, syrupi ferri iodidi, ½ to 1 teaspoonful in milk thrice daily, cod-liver oil, to which iodine may be advantageously added:

℞. Ol. morrhuae	100.00
Iodi purim	0.10

SIG.—One dessertspoonful or tablespoonful thrice daily.

and maltose, ferratose, etc.

7. SYPHILIS.

Etiology.—*Laryngeal syphilis,* on the whole, comes under our observation much less frequently than syphilis of the throat and mouth. It is, doubtless, very often overlooked in general practice, because the patients feel so little troubled in the milder cases that they do not consult a doctor, and because the physician himself—general practitioner or specialist for venereal diseases—not infrequently omits the somewhat troublesome laryngeal examination, more especially if the patient does not complain of his larynx or trachea, as is so often the case. Possibly, also, the laryngeal examination has been done at a time when all manifestations in the larynx had already subsided as the result of an efficacious general treatment. This may also explain the great differences in the statistical reports of the frequency of laryngeal syphilis, which vary between 0.4 to 30 per cent. I make it a matter of routine to investigate every case of syphilis with due regard to the laryngeal conditions, and have found that *the larynx is not often affected,* viz., in about 1.5 per cent. of all the cases. In the hospitals, which certainly receive the more severe cases, the percentage with regard to laryngeal syphilis might be different. How long it is after the infection has taken place that the larynx becomes affected is not easy to say, nor what the special cause may be which produces the laryngeal affection. The so-called secondary symptoms may occur two to three months after the infection, while the severer tertiary symptoms may appear sometimes even after the lapse of ten years and more—usually, however, especially under unfavourable conditions, after a few years. The predisposition to become affected is fostered by any of the chronic or acute irritations which are prone to diminish the power of local resistance. In some cases the disease is continued directly from the mouth and throat into the larynx. This is perhaps why syphilis mostly occurs on the epiglottis.

Tracheal syphilis follows on that of the larynx, but may also occur separately. Frequently, however, one does not see the patients until they have acquired tracheal stenosis from gummatous ulceration. (See p. 410.)

Symptoms of *hereditary* syphilis in the trachea or larynx are seemingly very rare.

Symptoms.—The anatomical changes and clinical symptoms, as one would expect, especially with regard to their development and retrogression, are analogous to those found on the mucous membrane of the mouth, throat, and nose. The *primary sore*, it is true, has not yet been observed in the larynx, although it might possibly occur through infection by unclean instruments. Laryngeal syphilis consists rather of the *secondary* and *tertiary* groups of symptoms, which, however, cannot be sharply separated one from the other.

(*a*) *Secondary syphilitic symptoms* are characterised by erythema or condyloma.

The erythema, which is usually associated with or corresponds to that found in the pharynx (see p. 312), shows the signs of a simple catarrh, accompanied by more or less secretion. The voice is hoarse or impure. It is irrelevant whether one classifies the condition as either erythema or catarrh.

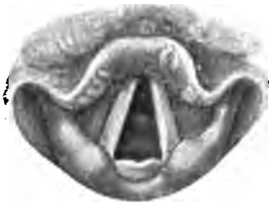

Fig. 177.—Condylomata of epiglottis (*Chiari*).

The condyloma (see Fig. 177) occurs in the form of mucous patches ("plaques muqueuses"), especially on the margins of the epiglottis, in the center of the vocal cords, and also on the posterior laryngeal wall. They resemble those "plaques" found on the oral or pharyngeal mucous membrane, and also form white, hoarfrost-like patches, which may sometimes be slightly elevated and surrounded by a reddened zone. The various irritations to which the larynx is exposed lead to desquamation of the epithelium and subsequently to erosions, which, if situated on one vocal cord, occasion a symmetrical affection of the other vocal cord by mere contact. This is a process identical with that seen on the skin, where a discharging papule is situated between two opposing folds—as, for instance, in the groin. In due course the small round-celled infiltration—the histological conformation of the condyloma—breaks down and superficial ulcers are formed.

The subjective symptoms are insignificant, and consist chiefly of disturbances of the voice, ranging from slight hoarseness to complete aphonia.

(*b*) *Tertiary symptoms* in the larynx consist histologically of excessive round-celled infiltration, either of a diffuse or more or less circumscribed character, by the breaking down of which the "infiltration ulcers" are formed. They may be distinguished as follows:

1. *Gummatous infiltration.* 2. *Gumma.* 3. *Gummatous ulceration.* 4. *Perichondritis and chondritis,* which, as in tuberculosis, might be produced by secondary infection or perhaps by the direct effect of the specific toxin. 5. *Formation of scar tissue.*

1. *The gummatous infiltration,* on laryngoscopic examination, appears as a diffuse, red, glistening, cushion-shaped thickening, which gradually merges into the adjacent tissues, and lends to the affected part a thickened, clumsy, and disfigured appearance, and tends often to very much narrow the lumen of the larynx. (See Figs. 178 and 179.) The infiltration may be found on all sides, but mostly, however, on the epiglottis and on the subglottic region, and is often accompanied by oedema of the neighbouring tissue. It does not usually cause much discomfort to the patient other than a certain amount of vocal disturbance, and subsides under proper treatment or even spontaneously. In severe cases, on the other hand, it might cause dyspnoea, and even occasion pain if actively ulcerating.

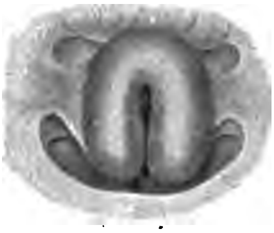

Fig. 178.—Gummatous infiltration of epiglottis (*Türck*).

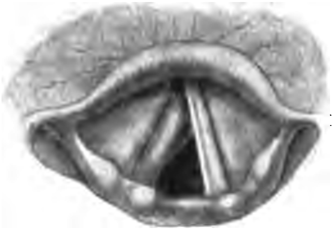

Fig. 179.—Gummatous infiltration of right vocal cord (*Chiari*).

2. *The gumma,* seated chiefly in the submucous tissue, is not very frequently met with in the larynx, and occurs singly or in groups.

It protrudes more or less, pushing the mucous membrane before it, and is of a dusky red, almost livid colour, and of a hard consistency. The gumma may subside after growing for a certain time, and, in such a case, the gumma shrinks in the center, forming there a dimple. In other cases, and more frequently, it softens in the center, and the softening process soon reaches the surface and gives rise to the formation of an ulcer, the edges of which are everted. Before the ulceration takes place the gumma assumes a yellowish colour. As has been already said, the seat of predilection for a laryngeal gumma is the epiglottis.

3. *The gummatous ulcer* (see Figs. 180 and 181) is the most frequent syphilitic manifestation met with in the larynx, owing to the fact that the infiltration rapidly breaks down. As in other places, so also here, the gummatous ulcer generally shows sharply cut, stiff, and indurated edges, deep, crater-shaped depressions, and a floor covered with a slimy film. The surrounding tissue is mostly, but not always, red and swollen, and may even be oedematous. Owing to the tendency of the gummatous ulcer to penetrate in depth, and at the same time causing little or no pain, extensive destruction and complications may ensue, which are liable to endanger life or, at least, to produce severe functional disturbances if the process is not arrested.

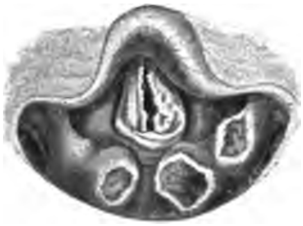

Fig. 180.—Gummatous ulcers on the vocal cords and aryepiglottic folds (*Türck*).

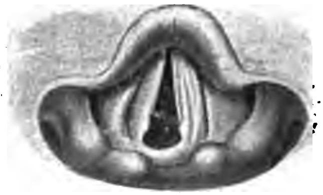

Fig. 181.—The same case as in Fig. 180 after healing up (*Türck*).

4. *Perichondritis and chondritis* are caused by cocci which gain entrance through ulcerations and have advanced into the deeper tissues. This method of origin is not truly specific in nature, and has already been discussed elsewhere. (See pp.

459 and 479.) It happens, however, that perichondritis developes without previous ulceration, that is, so to speak, ab initio, on the basis of a deeply situated and softened infiltration, and then makes its appearance on the surface. In my opinion it is not improbable that in such a case small superficial erosions have been overlooked. Certainly the fact that perichondritic symptoms do disappear after the use of iodides would seem to point to the specific character of the process. The seat of predilection of syphilitic perichondritis is the cricoid plate and the arytenoid cartilage. As regards the laryngoscopic appearances and complications, see chapter on Perichondritis Laryngea (p. 458, et seq.). (See Fig. 182.)

5. *Scar tissue* following on syphilis may be very considerable, owing to the deep and extensive defects occurring in the course of gummatous syphilis. As syphilitic scars are especially liable to contract, severe disfigurement, stenosis, and distortion frequently occur, whereby the whole configuration is altered, and incurable functional disorders may result. (See also p. 409.)

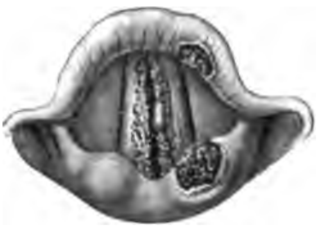

Fig. 182.—Both vocal cords are notched or serrated by gummatous ulcers; deep ulcer on left arytenoid; flat ulcer on epiglottis. Perichondritis arytenoidea duplex (*Schnitzler*).

It may be mentioned that in syphilis of the larynx paralysis of the vocal cords frequently occurs, and is due to either an infiltration of the muscles or to compression of the recurrent laryngeal nerve by infiltrated glands. Paralysis may be simulated by immobility of the vocal cords on account of spurious ankylosis of the laryngeal (crico-arytenoid) joint as the result of cicatricial contraction. In the trachea, mutatis mutandis, the same changes are met with; but here, still oftener than in the larynx, slight alterations are liable to be overlooked. The tracheal stenosis which is caused by the shrinkage of the scars left by gummatous ulcers, however, is so typical that they can hardly be mistaken. The ulcers are round, oval, grooved, or gutter-shaped, and may spread somewhat superficially. Usually, however, they penetrate in depth, and are prone to produce perichondritis or may even perforate into the mediastinum, the oesophagus, or erode one of the larger vessels. If syphilitic processes coexist simultaneously in the trachea and larynx, the gross anatomical changes are seen to be mostly in the upper part of the trachea; if, however, they are localised in the trachea only, then it is usually the bifurcation and the bronchi which are mostly affected (p. 410).

Diagnosis.—*Secondary* syphilis in the larynx and trachea is often so insignificant and frequently occasions so little discomfort that its specific nature, apart from the anamnesis, can be recognised only from the appearance of other symptoms of the skin and mucous membranes of a syphilitic nature, or by reaction to antisyphilitic treatment. *Tertiary* symptoms are liable to pass unrecognised, or to be mistaken for tuberculosis, more particularly if the anamnesis and the examination of the rest of the body give a negative result. It is true, however, that syphilis causes fewer subjective symptoms, but is, nevertheless, the more acute process, for syphilitic infiltrations break down sooner and more rapidly and predispose the adjacent tissue to become inflamed. But it is very difficult in any given case to arrive at a correct opinion as to the duration of the course of the disease. Very frequently, as, for example, in anaemic or debilitated patients, every sign of inflammatory reddening around the ulcer or infiltration may be altogether missing. Other signs also are often deceptive, and it is sometimes impossible, as has already been pointed out in the chapter on Tuberculosis (p. 488), to arrive at an exact conclusion as to the real nature of the disease, except by most circuitous methods. This is still more pertinent in diseases of the trachea, where the examination is so much more difficult than that of the larynx. In some cases direct examination of the trachea or bronchi may help, but this must always be very carefully done, as bronchial or laryngeal catarrh is nearly always present. Again, we wish to emphasise and to remind the reader that strictures in the larynx and trachea are chiefly due to contraction of the scars left by syphilitic ulcers.

The diagnosis becomes still more difficult if syphilis and tuberculosis are combined.

A distinction between syphilis and carcinoma may not be easily made by a laryngeal examination. In such a case a probationary excision of a small portion of tissue for a microscopical examination may be resorted to, and, on the other hand, a trial with iodide of potassium may be necessary.

The diagnosis of *hereditary syphilis* in the larynx cannot be made if no other characteristic signs of hereditary syphilis are present in other parts of the body, *e. g.*, the typical excavation of the upper incisors, interstitial keratitis, etc.

Prognosis.—The prognosis of laryngeal and tracheal syphilis, ignoring some few fulminating cases, is generally favourable, provided that the patient submits to treatment at an early stage. Unfortunately, *sit venia verbo,* the subjective symptoms are so little pronounced at first that the disease is usually already advanced beyond the stage of complete restitution before the patient goes to consult a physician. Infiltrations and gummata are prone to subside without leaving any consequences behind if they are treated properly and sufficiently early. If, however, ulcers have once developed, they do not heal without the formation of scars and defects, and it is just from these sequelae that irremediable disturbances or lesions of respiration, voice, and swallowing are set on foot, which may even become dangerous to life. In this sense the gummatous ulcerations of the trachea are especially ominous.

Treatment.—In any case where one can expect recovery, antisyphilitic treatment should at once be undertaken on the principles laid down in Part I. (See p. 104 et seq.) The general treatment may be successfully aided by local treatment, inasmuch as it may be necessary to incise a perichondritic abscess or to remove the necrosed part of a cartilage, or to perform tracheotomy, etc. Local treatment, however, must be made to take a secondary place to general treatment. On the other hand, local treatment is important, in cases of stricture and other sequelae, though it may not often be successful. (See Chapter on Strictures, p. 408, et seq.)

With respect to *rest* and *silence*, the same rules apply here as in tuberculosis.

8. SCLEROMA (LARYNGEAL SCLEROMA).

Etiology.—Scleroma of the larynx is very seldom primary. It usually follows the analogous disease of the nose and pharynx, and the trachea is almost always affected by way of the larynx. With regard to the occurrence of the disease and the rôle the scleroma bacillus plays in its etiology see Part I, p. 105.

Pathology.—Scleroma begins most commonly *below the vocal cords* as an inflammatory infiltration, usually of both sides, and shows the typical picture of hypertrophic subglottic laryngitis. From here it may spread downwards along the trachea, even into the bronchi, but oftener upwards to the ventricular folds, arytenoid cartilage, aryepiglottic folds, and epiglottis. There, pad-shaped or smaller nodular infiltrations of a pinkish or bright colour, and of a hard consistency, are seen to be developing. They seldom ulcerate, but change into a kind of fibroid tissue, which then shrinks, and so leads to the formation of cicatricial tissue and subsequently to stenoses.

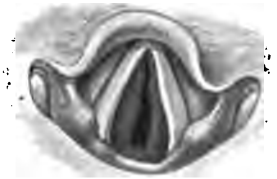

Fig. 183.—Scleroma laryngis. Subglottic laryngitis; subglottic pads (*Türck*).

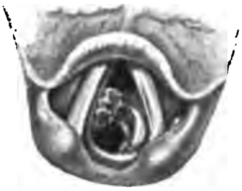

Fig. 184.—Subglottic knot of scleroma on the right vocal cord. Scythe-shaped scar on the right side of the trachea.

Symptoms and Course. (See Figs. 183, 184.)—Owing to the slow progress, the patients are at first not much disturbed, at least as far as the larynx is concerned. They may merely complain of some slight hoarseness and troublesome cough, due to the inspissated secretions forming crusts in the larynx. These crusts or scabs have an offensive odour, just as in ozaena. Later on, if the cicatricial contraction continues and the stenosis becomes narrower, then dyspnoea sets in, being greatly increased by the formation of crusts.

On laryngoscopic examination, large subglottic pads are found which sometimes narrow the rima glottidis to a small chink, or hole, if adhesions are formed at the anterior or posterior commissure. On the vocal cords and below them one may see grey or black crusts. If these crusts have been removed in one way or the other, then the mucous membrane appears pale and studded with nodules. The border-line between the vocal cords and the subglottic pads is, as a rule, marked out by a more or less distinct groove. The scars which develop from the infiltrations through contraction of the fibroid tissue show the most varied appearances, according to their seat and extension. The lumen of the larynx is altered thereby in a most extraordinary manner, and the picture becomes still more complicated if all other parts are infiltrated or if oedema is added, on account of disturbances of the circulation. In only exceptional cases would it be possible to gain a view into the trachea by ordinary laryngoscopy. Sometimes direct tracheoscopy will be more successful.

Diagnosis.—The coincident disease of the nose, throat, and larynx; the exceedingly slow and painless progress; the somewhat peculiar odour of the scabs; the conspicuous induration of the pale infiltrations; and, lastly, the absence of ulceration, point unmistakably to scleroma, and are quite distinct from any syphilitic disease. If one happens to be in doubt, iodide of potassium and the presence of the scleroma bacilli will afford proof.

We may again emphasise that subglottic swellings and pads are met with in some forms of chronic laryngitis. Possibly this form of chronic subglottic laryngitis, as also *Stoerk's* blennorrhoea of the larynx, is identical with scleroma. (See p. 448.)

Prognosis.—Although the disease must be considered as incurable, prognosis, nevertheless, is only bad when the process has advanced so far as to affect the trachea and bronchi. Scleroma advances very slowly and preserves its local character for a long time, so that it causes very little trouble except from dyspnoea, and the patients, moreover, are little disturbed, as it is painless. In some cases, perhaps, partially due to proper treatment, it was observed to have been altogether arrested and the infiltrations to have subsided. This, however, very seldom occurs, and fresh recrudescences are the rule. In severe stenosis a sudden accumulation of secretion may cause death from suffocation.

Treatment.—The possibility of influencing scleroma by *Röntgen* rays or radium has been already alluded to in the discussion of rhinoscleroma. For the rest, the treatment can only be symptomatic and must aim at the mitigation of the troubles ultimately ensuing. Crusts and scabs may be removed by inhalations or instillations, and stenosis should be treated by *O'Dwyer's* or *Schrötter's* methods. (See pp. 422 and 429.) It has been observed that, by the pressure exercised by the tubes inserted, the infiltrations have been made to subside if the process was not too long standing. Cicatrices must be systematically stretched. This treatment ought to be continued at intervals for years, and it will not occasion any difficulty for the patients, as they finally learn themselves to manage the insertion of *Schrötter's* vulcanite tubes. The results of treatment are much less favourable if the trachea has been the affected part. If, in such a case, the affected parts cannot be reached through the mouth, then tracheotomy must be performed, and the stenosed part must be dilated from the tracheotomy wound in the manner before described. (See p. 429.) Tracheotomy should also be done if there is any danger of suffocation. Afterwards, dilatation with tubes and bolts, after the manner of *Schrötter* (see p. 429), must be undertaken.

9. MALLEUS (GLANDERS).

The changes caused by malleus in the larynx very much resemble those found in glanders of the nose and pharynx. (See pp. 107 and 315.) They consist of ulcers originating in infiltrations, and which look very much like tuberculous ulcers. Laryngoscopic investigations have not yet been published.

10. LEPRA (LEPROSY).

The larynx is affected in all cases of leprosy, though sometimes late in the disease. It begins with all the symptoms of a simple catarrh, but after a time diffuse or circumscribed infiltrations appear. The epiglottis is mostly, often alone, affected; much later on, however, the other portions of the aditus laryngis become involved. The epiglottis is altered in shape, becomes thickened, and assumes the form of an omega, Ω; this, in combination with the thickened aryepiglottic folds, renders laryngoscopy difficult. Later on circumscribed infiltrations occur, and then resemble very much the nodes of lupus. The infiltrations break down and then form smaller or larger ulcers, which destroy the tissues, especially the epiglottis. Or they form white, glistening scars, which lead to serious adhesions and stenosis. The deformity may assume such a degree that all orientation may be lost.

Symptoms.—*The subjective symptoms* are dependent on the site and the extent of the changes. The voice becomes impaired, hoarse, or aphonic. There is a sensation of burning and tickling in the throat, sometimes so aggravated as to cause coughing fits, and later on dyspnoea becomes predominant. There is no pain, on account of the anaesthesia of the mucous membrane. This anaesthesia, which is also pronounced in the pharynx, renders laryngoscopy a comparatively easy matter.

Diagnosis.—The diagnosis should offer no difficulties if the whole syndrome of symptoms, both intralaryngeal and extralaryngeal, is taken into consideration, for the remarkable anaesthesia of the mucous membrane is typical of leprosy. In doubtful cases the bacilli of leprosy should be looked for.

Prognosis.—Although the patients themselves are resigned to the slowly developing dyspnoea, still the prognosis is very bad, for in the long run stenosis claims its victim, if an intercurrent disease or general exhaustion does not put a premature end to the patient.

Treatment.—This can only be symptomatic, and troublesome disorders, such as dyspnoea, may be relieved by tracheotomy, etc.

VII. FOREIGN BODIES AND PARASITES.

1. FOREIGN BODIES.

Etiology.—Foreign bodies nearly always reach the larynx by way of the mouth, and are then mostly aspirated. The aspirated objects may be derived from outside or inside the body. In the latter case it is usually blood or vomited matter which has been drawn into the larynx during general anaesthesia. Sometimes, however, a portion of a necrosed cartilage in perichondritis might be caught between the glottis during coughing, or an excised pharyngeal tonsil falls down into the throat and is aspirated. Occasionally, also, a calcareous bronchial gland perforates a bronchus and becomes in this way a foreign body; but even accumulated and non-expectorated secretions also may become thickened and thus constitute a foreign body.

Objects derived from outside the body are aspirated while speaking, laughing, or yawning during eating. It is a bad habit to hold needles, buttons, etc., between the teeth. A sudden fright, which also causes a deep inspiration, might be the cause of aspirating a foreign body.

In children and lunatics very curious objects sometimes find their way into the larynx. Semiconscious or comatose persons, who are no longer able to swallow correctly, on account of reduced reflex irritability, are liable to aspirate food-stuffs if they are not very carefully fed. Artificial teeth, which have become loose during sleep or narcosis, play a great rôle in the larynx among foreign bodies. It has occasionally happened that a swab, brush, or part of a broken instrument has been left behind after an operation.

A foreign body does not often reach the larynx through a tracheotomy wound, and, still more rarely, through wounds caused by violence to the larynx or trachea.

Pathology and Symptoms.—If a foreign body has gained access to the larynx, then, owing to the intense sensitiveness of the mucous membrane, it at once excites reflex coughing, and by this small foreign bodies, such as particles of food, may be brought up again, leaving nothing behind but a mere congestive hyperaemia which soon disappears. Although the foreign body may have rested for a moderate period in the larynx, the subsequent irritative symptoms need not be very severe. But if the foreign body lodges for a longer time in the larynx, then symptoms of irritation will not be missing, and the reactionary changes caused by it run through the whole scale from the mildest to the greatest degree.

(*a*) **Foreign Bodies in the Larynx.**—Larger sized foreign bodies are arrested in the larynx for the reason that the larynx is evenly narrowed from the aditus to the glottis, and the glottis itself closes firmly by reflex action. If the foreign bodies are large enough to fill the laryngeal cavity, death from suffocation will very quickly ensue. The epiglottis also, if depressed by a large bolus, may play a very dangerous rôle. (See p. 318.) If the foreign body is of small size, it may, nevertheless, cause acute obstruction: the patient struggles for breath, becomes cyanotic, coughs, chokes, or vomits, but the attack, however alarming, passes away, and the patient becomes quiet again. If the mucous membrane has been injured by the foreign body, the material which is brought up is blood-stained; there are indefinite pain and hoarseness, or even aphonia if the vocal cords are impeded in their movements by the foreign body or by swelling of the soft parts. Occasionally these symptoms are very indefinite, especially if the sensibility of the mucous membrane was already diminished or if the foreign body is so small that it has slipped into a corner or fold of the mucous membrane, *e. g.*, into the ventricle of *Morgagni.* One must, however, be prepared for a small foreign body, which is not actually impacted, to change its position and so cause severe trouble and even danger of suffocation. In some cases, by its movements, real coughing paroxysms are produced which very much resemble whooping-cough, or the patient himself feels the movement during respiration and coughing.

(*b*) **Foreign Bodies in the Trachea and Bronchi.**—Small and round foreign bodies more easily slip downward through the glottis than larger and more uneven ones. If, however, long splinters of bone, coins, etc., pass into the trachea or bronchi, it is due to the circumstance that they have passed the chink between the vocal cords in a position favourable to their gliding through "end on." If a needle, fishbone, splinter, or other pointed and long body has reached the trachea, it is usually caught somewhere in the mucous membrane and does not cause alarming symptoms. The mucous membrane of the trachea is supposed to be more tolerant than that of the larynx. But here, as well as in the larynx, the symptoms may be very acute. Of the two bronchi, it is the right one which is oftener occupied by a foreign body (see p. 358), for the reason that it is the straighter and wider of the two, and, as is supposed also, from the greater power of suction exercised by the right lung than by the left. If the foreign body remains movable, it may happen that it is again flung out by coughing, and the impact against the closing vocal cords is perceived by the patient himself as a peculiar slapping sound.

As has been already said, foreign bodies may occasionally reach the trachea through a tracheotomy wound, *e. g.*, a fragment broken off a worn-out canula.

Sequelae and complications of foreign bodies in the air-passages depend on the size, shape, and site of the body, and also on whether it is infective, and how long it remains in situ. A foreign body which has lodged for a lengthy period provokes symptoms of irritation, which are the more severe the longer it has remained in situ, and the more the mucous membrane has been irritated by the uneven surface of its edges, points, etc. It sets up an inflammatory swelling of the surrounding soft parts, tending to produce erosion or local necrosis, and subsequently leading to ulceration and granulation. Phlegmonous inflammation may even take place, which not only spreads into the adjacent tissues, but may also endanger life by threatening suffocation. In some cases cutaneous emphysema has been observed. The ulceration may also lead to perichondritis or to serious perforation of the trachea, and finally the granulations, through the formation of connective tissue, may give rise to contracting cicatricial tissue, with subsequent stricture, as has been already mentioned. The corpus delicti may actually become encapsulated in granulations or scar tissue and thus become concealed. Not infrequently needles wander and appear on the surface of the body or somewhere in the thorax.

If the foreign body is lodged in a bronchus, it might either, by itself or by accumulation of blood, mucus, or pus, cause obstruction and atelectasis of the corresponding lung. In other cases pneumonia may occur, following infection by the foreign body or ulceration leading to perforation of the bronchial wall, or the inflammation may spread further into the tissue of the lung, causing abscess or gangrene. In a postmortem of such a case the foreign body may be found in a cavity of the lung. Sometimes, also, the mediastinum, pleura, and pericardium may be implicated.

Diagnosis is based on: (1) Anamnesis; (2) clinical and subjective symptoms; (3) objective symptoms, as seen on laryngoscopy, tracheoscopy, and examination of the bronchi.

1. *Anamnesis.*—In many cases the patient or his friends report the aspiration of a foreign body; for this reason one should never forget to inquire. Unfortunately, the anamnesis does not help in the case of small children or of adults who have been unconscious at the time of the accident. Moreover, the reports are often confused, and not infrequently admit of doubt as to whether the foreign body has entered the air-passages or the gullet.

2. *The clinical or subjective symptoms,* after the first attack has abated, are not very characteristic or may be altogether absent; if, as has been pointed out above, consciousness or the sensitiveness of the mucous membrane was in abeyance or suspended at the time of the mishap, more especially so if the foreign body becomes engaged at a spot where it does not cause any marked disturbance. On the other hand, we must reckon with all sorts of paraesthesiae, particularly in excitable and nervous patients (see p. 318), which may have arisen from the presence of a foreign body which may have already been coughed up and expelled, the cough being excited by slight irritations left behind. Anyway in children who are prone to suffer from coughing paroxysms or fits of choking, not only whooping-cough and diphtheria, but also the possibility of the presence of a foreign body, should be borne in mind. Among other signs which would help to aid diagnosis may here again be mentioned the peculiar slapping or clattering sounds accompanying the foreign body if it happens to be lodged in the bronchi, and also that sometimes there may be an inspiratory and expiratory bruit or vibration, which may be distinctly felt. In some cases the patient feels some pain at a particular spot, a valuable sign,

which is, however, often deceptive, in that the patient's power of localising his pain is, as has been said, very often erroneous.

If we suspect the foreign body to be in the bronchi, then examination *of the lung should never be omitted.* (See chapter on Stenoses, p. 408.) It is true that if the foreign body has been arrested in a bronchus for a length of time, and the signs in that particular lung should point to severe bronchitis, bronchiectasis, or even phthisis, these would in nowise help us further. But taken together with the history, the subjective symptoms, and perhaps the positive result by transillumination with *Röntgen* rays, would eventually assure a diagnosis, and informs us sufficiently concerning the extent and intensity of the secondary complications.

3. *The direct examination* for a foreign body by *means of mirror and tubes* is, of course, of the highest importance, though we may here often meet with many difficulties. Simple laryngoscopy or *Kirstein's* method of autoscopy may at first be applied, in order to inspect the larynx and the trachea as far down as possible; and if we have not satisfied ourselves, we could then perform tracheoscopy and bronchoscopy by the direct methods.

Examination by means of the mirror is very difficult in restive and excitable persons, and it is, moreover, *not reliable* for transparent objects and those which have completely, or almost so, embedded themselves. The resistance on the part of the patient might be overcome by local anaesthesia, and in children, superficial general anaesthesia with *Kirstein's* autoscopy will perhaps succeed. We must, nevertheless, remember that the foreign body, by the time of examination, may have already become enveloped in mucus and secretion, and therefore be hidden from direct observation. In such an event a localised swelling and redness should arouse our suspicion as to the presence of a foreign body. Accumulated mucus must be wiped away by means of swabs or aspirated by a syringe. It very often happens that the first examination is negative, and this being so, and if there is no imminent danger or great discomfort, we may confidently wait until the patient has become calmer.

If the larynx has been found empty, if nothing has been discovered in the trachea, if we did not succeed by the laryngoscope with one or other method, and if we still have reason to assume that there is a foreign body, probably in the trachea or bronchi, then *Killian's* method of direct tracheoscopy (bronchoscopy) must be resorted to, either through the mouth, or if tracheotomy had been necessary on account of any danger of suffocation, then through the wound.

The diagnosis may be greatly assisted by *transillumination* and by *skiagraphy by means of the Röntgen rays*; chiefly, however, where metallic or osseous objects are concerned. If a skiagram be taken from various directions, then the foreign body may be exactly located, and its distance from the thoracic wall measured. This would naturally be of enormous value if operation be intended. If the foreign body has been discovered, it will be well to come to a conclusion as to its size, site, and shape; for we will have to direct our methods accordingly. It is a well-known fact that one very often mistakes the size of the object, as it is usually supposed to be much smaller than it actually turns out to be.

Prognosis.—A foreign body of itself rarely menaces life, as it would have to be very large, or the larynx very small, for it at once to cause death by suffocation. On the other hand, the secondary symptoms determine the course of events. The prognosis, therefore, is best when the foreign body has been quickly removed. In this respect foreign bodies in the larynx—other things being equal—are less serious than those in the trachea or bronchi. In children a foreign body soonest leads to obstruction or stenosis, on account of the natural smallness of the air-passages, and, also owing to the greater difficulty in examination which we encounter. The chances are somewhat more unfavourable.

Treatment.—A foreign body having been discovered, it should be removed in the quickest way possible. This commandment may be obeyed in various manners. If it has been merely misswallowed, *i. e.*, if a particle of food or a drop of drink has "gone the wrong way," then it is quite sufficient to slap the patient's back, or to quiet him and make him drink a mouthful of water several times. It is, however, not usual to call in a physician for such an episode, as usually his aid will only be required if the foreign body has become lodged in the air-passage for a shorter or longer time. Then our proceedings must be guided by the circumstances, according to whether or not there is imminent danger.

1. *In threatening suffocation,* one must try to seize the object with the finger, if one suspects it to be in the aditus; *e. g.*, a morsel of food. Or if one were sure that a small round object, such as a bead or pea, or small button, etc., has been aspirated, then the patient could be reversed into a position with the head hanging down, and then required to forcibly cough while in this position, and at the same time be slapped on the back. If this procedure does not quickly succeed in dislodging and ejecting the foreign body, then tracheotomy must be performed. Unfortunately, it often happens that one is called in to an already moribund patient. Setting aside such an occurrence, an operation will at once serve to restore free respiration, for if the foreign body is retained in the upper part of the air-passage, it will be easy at the same time to extract it through the wound, as the foreign body will not usually be far away from it. It not infrequently happens, however, that the foreign body is flung out with the cough excited by the opening of the trachea; or if it had been impacted, it will have become so much loosened that it may then be easily removed.

Foreign bodies seated *above the tracheal wound* should be pushed upwards without using any force, and then extracted through the mouth (see below).

If the foreign body, however, is seated below the tracheal wound, one must be speedy with further procedures. A probe or tube, such as is used for direct bronchoscopy (see p. 382), can be inserted, and the body seized by means of forceps, such as are devised by *Killian,* and which can be modified according to the site, consistence, and shape of the body. (See Fig. 185.) If the body cannot thus be extracted through the tube, then sometimes one succeeds by pulling it against the end of the tube, and so be able to remove it on withdrawing the tube.

2. *If there is no imminent danger*, then extraction or removal per vias naturales should be attempted. If the foreign body

is found in the larynx, it may be removed with the guidance of the laryngoscope, and if in trachea or bronchi, with the aid of direct tracheoscopy or bronchoscopy. If the foreign body should be in the uppermost part of the trachea, then the laryngeal mirror will be sufficient. In any circumstance local anaesthesia will be necessary, and it is, perhaps, also advisable to apply adrenalin or renoform in order to render the foreign body more plainly visible on account of the shrinking of the mucous membrane so produced.

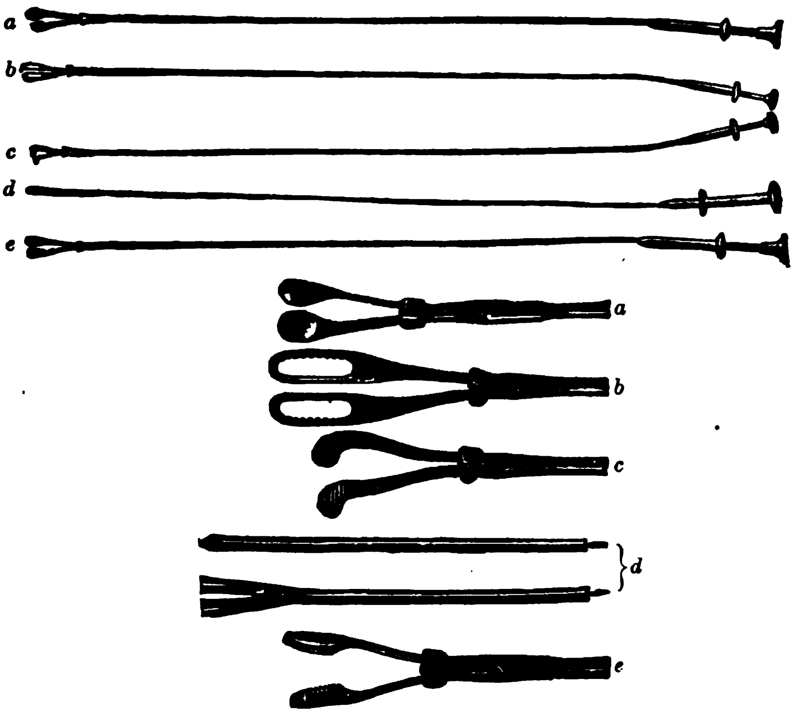

Fig. 185.—Instruments for removal of foreign bodies (after *Killian*): (*a*) Spoon forceps; (*b*) forceps for the removal of beans; (*c*) forceps for needles; (*d*) separating forceps (opened and shut); (*e*) tweezers.

Before commencing to extract a foreign body, consider well which instrument you will use and how to manage it. It is just here that unsuitable and rash manipulations so often have dangerous consequences, either by unnecessarily and injudiciously injuring the mucous membrane or they might, by shifting or displacing the body, even cause serious dyspnoea, which will necessitate tracheotomy. That the extraction of a pointed or impacted body is often very difficult goes without saying. If one is not able to seize and remove it by rotatory or levering movements, it must be broken up and extracted piecemeal, that is, if its consistency allows such a proceeding. No hard-and-fast rule can be given: each case will have its own peculiarities.

Generally speaking, round smooth objects, which are liable to slip away, may be seized with the spoon forceps, and pointed or lengthy objects with pincers or similar instruments. Hollow bodies may be removed with separating instruments, which can be closed and inserted into the lumen of the body, and then opened, so as to seize it. (See Fig. 185.)

For some cases fine hooklets are appropriate.

In the case of children the difficulties are still more increased, as in them nothing but general narcosis may be of assistance, along with the application of autoscopy.

We cannot warn the operator sufficiently *against using force* during the extraction. If there is no great hurry, one may pause for a while. It does happen occasionally that the foreign body, which has been loosened by the attempts at extraction, is subsequently coughed out spontaneously.

If all attempts at extraction have failed, with and without tracheotomy, we must resort to laryngotomy (laryngofissure) for foreign bodies lodged in the larynx; and if such is seated in a bronchus, then pneumotomy will be required; but the latter, only in cases where the examination by x-rays has given us reliable information.

2. PARASITES.

Animal parasites do not often gain access to the larynx or trachea. Sometimes, however, small insects find their way into it when drawn in with a strong breath, but they scarcely find their new surroundings congenial. Leeches have occasionally found their way into the larynx from impure water or from the mouth, where they may have been applied for some medical purpose. It may be mentioned that trichinae rather prefer the laryngeal muscle in which to encapsulate, and also that ascarides occasionally crawl into the larynx or trachea.

Vegetable parasites are also of rare occurrence in the air-passages. Thrush has been observed in neglected cases to fungate from the pharynx downwards into the larynx. (See p. 212.) One sometimes finds the patches of "thrush" fungi lining the trachea as far down as the bronchi, showing that "thrush" also grows on other than squamous epithelium. Some authors, among them *Bukofzer*, have described a primary "thrush" in the larynx. In all these cases diagnosis is not difficult if the mouth and throat also show the same affection. Otherwise microscopic examination will serve to clear up the matter.

Occasionally leptothrix, and in one case of *Hindenlang* a fungus called Pleospora herbarum, has been described. The latter fungus usually grows on certain herbs. Actinomycosis of the larynx, inasmuch as one can judge from the few cases published up to the present, is mostly secondary to that of the mouth or throat. Swellings occur, which are either outside, occupying the thyroid or cricoid cartilage, or are seated inside the larynx, thus bulging forwards the mucous membrane, where they may be easily mistaken for new-growths. In such a case the diagnosis can be made only by a microscopic examination of an excised piece; or even not until after an operation, if it be that one was unable to find any similar actinomycotic disease in the region of the parotid, which might guide one as to the nature of the laryngeal affection.

VIII. INJURIES.

The larynx and trachea are injured by agencies acting either from without or from within. In the former case the injuries are characterised as contusions, wounds, fractures, dislocations, etc.; in the latter, as cauterisation, scalding or burning, or operative but unintentional lesions, and, as *Hopmann* terms it, as haemorrhage and rupture due to overstraining of the muscles. On the whole, such injuries are not very frequent apart from injuries inflicted with suicidal intention.

A. EXTERNAL INJURIES.

1. **Contusions** are sometimes caused by a blow, thrust, or fall upon the neck. The symptoms usually correspond to the force exercised. There may be pain on speaking and swallowing, hoarseness, and aphonia. On laryngoscopic examination one finds redness and swelling, ecchymosis or free haemorrhage (so-called traumatic haematoma of the larynx). The neck may show a certain amount of rigidity. The treatment consists in keeping the larynx at rest and the application of ice. The course is mostly favourable, but may be complicated by oedema or abscess.

2. **Wounds.**—Gunshot wounds are mostly observed on the field of battle; punctured wounds, after a hand-to-hand struggle or attempt at assassination; incised wounds are almost exclusively observed in cases of committed or attempted suicide. In gunshot wounds the walls of the larynx are not often cleanly perforated; but much more frequent are complicated lacerations, accompanied also by the same effects in the neighbouring tissues. Punctured wounds do not usually bleed much, but, on the other hand, an extensive cutaneous emphysema, due to the air forcibly inspired and driven into the wound, is liable to compress the trachea and thus cause dyspnoea. Incised wounds commonly run across the neck, and, though the large vessels usually escape injury (see p. 259), yet cause serious haemorrhage. If more than the soft parts have been cut through, it is usually the thyroid cartilage that is concerned; but the incision, however, might be situated above it, through the hyothyroid ligament, or below it, through the trachea; and if great force was used, may extend to the vertebral column. The edges of the wound always gape considerably. If the trachea is wounded, aphonia is mostly present; otherwise hoarseness, dysphagia, and dyspnoea occur; the last especially, being caused by the swelling of the mucous membrane, collection of blood-clots, portions of cartilage which may have been separated, or even through subsequent perichondritis. Blood is likely to flow downwards into the trachea or bronchi, and causes paroxysms of coughing or even dangerous attacks of dyspnoea. Gunshot wounds are less favourable than incised

and punctured wounds, from the fact that as they heal strictures due to cicatricial tissue may ensue. Death may result from haemorrhage, suffocation, sepsis, or pneumonia.

Treatment.—All haemorrhage must be stopped and any danger of suffocation averted by tracheotomy, followed by the insertion of a *Trendelenburg's* tampon-canula. The wound must, of course, be treated according to surgical indications. Primary suture will not often be successfully applied, and for the rest, the treatment is mainly expectant. Stenoses are to be treated, if nothing occurs to interfere in the mean while, after complete recovery.

3. **Fractures,** like contusions, are caused by external but blunt force, *e. g.*, strangulation, blows, etc. Fracture becomes easier the older and more brittle the cartilage may be, on account of the normal ossifying processes; and for this reason fractures are seldom met with in young people. The thyroid cartilage is the most frequently fractured, owing to the fact that it ossifies comparatively early in life, and is freely exposed to external violence. Other cartilages are not often fractured; but perhaps because the injuries are oftener overlooked or mistaken for simple contusions. The line of fracture almost always runs vertically; comminuted fractures are rare, and are only caused by great violence, and then are usually associated with fracture of the hyoid bone.

Symptoms may be insignificant in slight cases, and, on the other hand, there may be local pain on swallowing, and also dyspnoea; and if the mucous membrane was also injured, then haemoptysis and emphysema may be present. The soft parts show swelling and oedema, and both may increase to such a degree that suffocation may threaten.

Diagnosis.—On inspection we notice a flattening or broadening of the cartilage; by palpation we may be able to ascertain displacement or abnormal mobility and crepitation. In some cases palpation is rendered difficult or impossible because of the tenderness and swelling of the parts concerned; but in others a perforating wound in the skin may facilitate the diagnosis. Laryngoscopically, we usually find much the same conditions as in contusion (see above). The laryngoscopic examination is usually less reliable as regards the actual fracture and its site, but more reliable with respect to the degree of the ensuing stenosis.

Prognosis is in any case doubtful, for although the fragments may unite by fibrous union, yet stenosis not infrequently results. Unfortunately, many cases end fatally in the first stages, through asphyxia, or death supervenes later from inflammatory oedema, wound infection, pneumonia, etc.

Treatment.—In slight injuries we may adopt an expectant line of treatment. In all other cases a prophylactic tracheotomy should be performed, so as to be protected and safe from unpleasant consequences. The reposition of the fragments may be attempted through the tracheotomy wound, by means of gently and carefully manipulating a catheter or probe, or chimney canula. But one must beware of unnecessary meddlesomeness, as little or no good will be achieved by it, and much harm may result. To control the swelling of the soft parts, ice should be applied externally and internally.

4. **Dislocations,** per se, are not often recognised. They are, as a rule, combined with other injuries, and mostly with fractures. Laryngoscopically, displacement and immobility of the arytenoid cartilages may be noticed, besides the usual signs of injury.

B. INTERNAL INJURIES.

1. **Cauterisation and Scalding.**—*Cauterisation* is usually the result of the action of strong acids or alkalis; *scalding* is caused by hot fluids or vapours. In the bulk of cases only the mouth, pharynx, and oesophagus are usually affected; but frequently, however, combined with such injuries are those of the aditus laryngis, due to the inhalation of steam at a high temperature. The lower regions of the air-passages may also be more or less severely injured. The lesions, if the injury was a slight one, consists of reddening and swelling of the mucous membrane. In the more severe cases, if the hot fluid or steam was in longer contact with the mucous membrane, then erosions or ulcerations occur, which sometimes show a fibrinous or diphtheritic exudation, with the formation of pseudomembranes, and are prone

to lead to phlegmonous inflammation, with extensive inflammatory oedema of the entrance of the larynx, caused probably by staphylococcal invasion. In such cases stricture is usually the result.

Treatment.—The diet must be regulated, and must consist of fluids or sloppy ingredients. Internally and externally ice should be applied, and menthol pastilles may be used or instillations of 10 per cent. to 20 per cent. of menthol in oil may have a good effect.

2. **Unintentional Operative Lesions.**—In restive persons intralaryngeal manipulations of all kinds (surgical and medical), and unskilled handling of the instruments, may be the cause of such injuries. Danger of suffocation might occur if blood from grosser injuries runs down into the trachea.

3. **Haemorrhage and rupture from muscular overstraining** sometimes occur in persons suffering from some irritation in the larynx, as the result of severe coughing, retching, or shouting. They give rise to haemorrhage beneath the mucous membrane (ecchymosis, haematoma), or also to free haemorrhage into the larynx, and subsequently to haemoptysis. Laryngoscopically, in such a case, one may sometimes be able to see the bleeding vessel, but not infrequently it may be discovered on the lower surface of the vocal cords. On the whole, one should not be in a hurry to diagnose free haemorrhage into the larynx without having good reasons for doing so, and one must always remember that free haemorrhage into the larynx is mostly derived from the lung or nose. (See pp. 378 and 390.)

Treatment consists in absolutely resting the larynx and in the application of ice; eventually, a sedative or narcotic may be ordered.

IX. TUMOURS.

Etiology and Pathology.—We do not know much concerning the origin of laryngeal or tracheal new-growths, which may be benign or malignant. We may be tempted to attribute the cause of tumours to every variety of chronic irritation, in just the same way as they are the source of chronic laryngeal catarrh;

and various authors, indeed, have ascribed to irritation the same etiological importance with regard to the development of new-growths. It is, however, remarkable that of the great number of cases of laryngeal catarrh, only a very few are associated with tumours; besides, it is quite impossible to say, in any given case, whether the laryngitis was the cause of the tumour, or the tumour the cause of the catarrh. One cannot help assuming that a certain predisposition, taken in conjunction with some local irritations or other causes, may give rise to the development of new-growths. We must also take into account, perhaps, this indefinite predisposition in the cases of benign tumours in small children, which, like papilloma, it would sometimes seem, occur congenitally.

Generally, the male sex shows a greater liability to laryngeal tumours than the female, and people of middle age are in this respect also more liable. Papilloma is of very frequent occurrence in children; while carcinoma, as is usual, occurs mostly in older people—mostly in those over forty years of age. Sarcoma is altogether rare, and does not admit of any general rule as to its frequency at different ages, though it is certain that it occurs oftener in younger people than does carcinoma. That a benign tumour may suddenly assume a malignant character cannot be denied, but, according to *Semon*, this is exceedingly rare.

A. BENIGN TUMOURS.

Pathology.—In the majority of cases these are fibrous tumours, viz., fibromata or papillary fibromata, otherwise known as papillomata. Cystic tumours are less frequent, and angioma, lipoma, myxoma, enchondroma, adenoma, amyloid and thyroid tumours, are all very rare.

1. **Fibroma.** (See Figs. 186, 187.)—It is a tumour of round, oval, or tuberous shape; either broadly sessile or provided with a pedicle, it is usually of a small size, from that of a pin's head to the size of a pea. Its surface is smooth, whitish or pink, and its consistency hard or somewhat soft. Fibroma is usually single, and is then situated in the middle or at the anterior end of a vocal cord. In one case under my own obser-

vation a papilloma of the size of a small pea was seated on the middle of the surface of the left vocal cord, whose margin was left free, and the papilloma swung to and fro at every movement, sometimes even across on to the right vocal cord, if the glottis was closed. (See Figs. 188, 189.) The growth of fibromata, as a rule, is very slow.

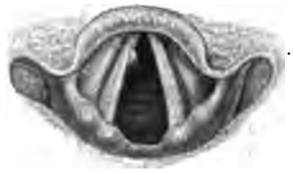

Fig. 186.—Fibroma of right vocal cord (*Türck*).

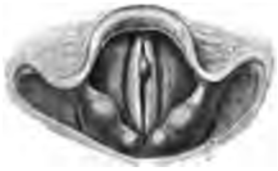

Fig. 187.—Same as in Fig. 186 during phonation (*Türck*).

A fibroma consists of connective tissue, between the bundles of which interstices or spaces are formed by serous imbibition (soft fibroma); or the bundles of connective tissue may lie close together and contain elastic fibers (hard or true fibroma). *Hard fibromata* in the larynx are rare, but they may grow to a large size, and are mostly found at the entrance of the larynx.

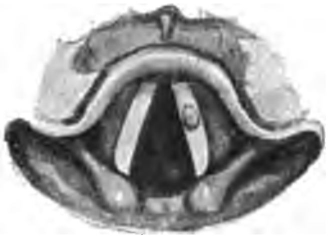

Fig. 188.—Pedunculated fibroma of left vocal-cord, arising from the upper surface.

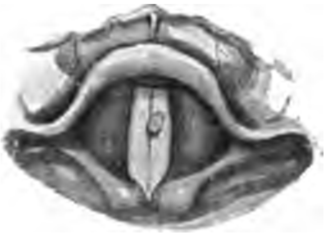

Fig. 189.—Same as in Fig. 188 during phonation.

The *soft fibromata* represent, with regard to their histological structure, circumscribed inflammatory products, as do also "singer's nodes" (see p. 449) and mucous polypi of the nose (see p. 114). They are only classed with the tumours on account of their clinical appearance. In the trachea fibromata are less frequently seen, and exactly resemble those found in the larynx.

2. **Papilloma** usually occurs multiple, seldom single; and varies much in size, shape, and site. It forms warty, cockscomb-like, grape-like, or cauliflower-shaped tumours, which show a white or pink colour, according to the thickness of the covering epithelium, and their own vascularity. Single warts or nodes cannot be distinguished from fibroma. This similarity is the reason why some authors think that papilloma occurs more frequently, and why others think the same of fibroma. Very often, however, both kinds of tumour may occur together. (See Figs. 190, 191, 192.)

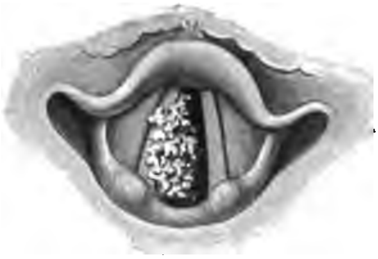

Fig. 190.—Papilloma of right vocal cord (*Oertel*)

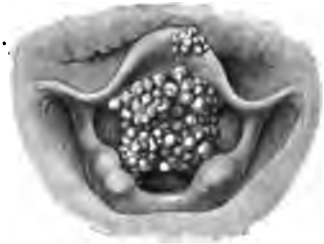

Fig. 191.—Multiple papillomata in larynx and on epiglottis (*Oertel*).

The seat of predilection is usually the vocal cord, but the papilloma may also be found on other parts of the larynx, *e. g.*, on the ventricular folds, epiglottis, and aryepiglottic folds, where they may grow so quickly and exuberantly that they may fill the entire lumen of the larynx as with one mass of cauliflower-like growth.

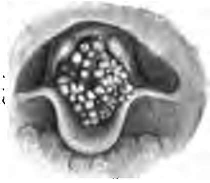

Fig. 192.—Excessive development of papilloma in a child (*Oertel*).

Apart from this characteristic appearance, two qualities of the papilloma distinguish it from all the other tumours, viz., they mostly occur in children, and possess a hideous disposition to recur.

Occasionally they undergo spontaneous retrogressive changes, as, for instance, after a tracheotomy performed on account of threatening suffocation or at puberty. Papilloma in adults is said to be the forerunner of a carcinoma slowly developing in the depths of the tissues.

Histologically, papilloma shows the same structure as fibroma, viz., connective tissue, of conical or papillary outline, covered with several layers of squamous epithelium, varying in thickness, and containing a greater or lesser number of blood-vessels. The external appearance of the tumour is dependent on the arrangement of its cones or papillae.

Tracheal papillomata occur jointly with those in the larynx, and are, so to speak, only a continuation downwards of the latter.

3. **Cysts.** (See Figs. 193, 194.)—Cysts are really not frequent in either the larynx or trachea. They form well-defined rounded or oval fluctuating tumours, from the size of a pin's head to that of a cherry. The mucous membrane covering the tumour is grey or greyish red, and shows a neat regularity of vascularisation. They are sessile or pedunculated, and occur mostly on the surface of the epiglottis or vocal cords, or they arise from *Morgagni's* ventricle. They represent true retention cysts, which develope in blocked and subsequently dilated glands; but sometimes they are congenital formations, the etiology of which is not quite clear (*Cohnheim's* theory of aberrated embryological cells; branchogenic cysts, etc.). Occasionally, traumatic cysts have been observed by *Chiari* and *Glas*. The small cysts and longitudinal or pleated formations occurring on the vocal cords are considered by *Schrötter* and *Chiari* to be inflammatory products which have their origin in a serous infiltration of a small hypertrophy of the marginal epithelium of the vocal cord.

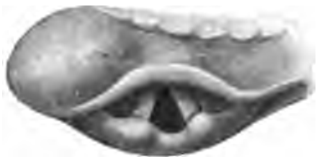

Fig. 193.—Cyst of the epiglottis (*Mackenzie*).

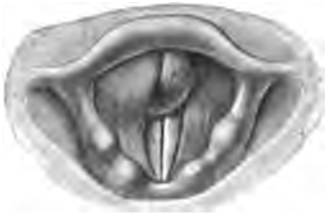

Fig. 194.—Cyst arising from the right ventricle (*Bruns*).

4. **Other benign tumours** occurring in the larynx and trachea

are of no practical importance, owing to their rare appearance.

Angioma is found on the vocal cords, forming mostly small, dark or brownish, lobulated or tuberous tumours, which consist of a network of dilated vessels or cavernous tissue. Histologically, they can be distinguished from the fibro-angioma, lymphangioma, and other mixed formations, which are not composed of newly formed blood-vessels, but are only conglomerations of dilated or ectatic blood-vessels.

Lipoma is a slow growing tumour, which may, however, attain a considerable size, and its seat is usually at the aditus laryngis.

Enchondroma forms pale, tuberous, flat tumours, of hard consistency, and arise mostly from the cricoid plate.

Thyroid tumours consist of tissue of the thyroid gland, and usually occur in conjunction with a goiter growing inwards between the laryngeal or tracheal cartilages. Such a tumour, if occurring, is usually found in the trachea, and is known as an intratracheal goiter (struma intratrachealis).

Amyloid tumours are multiple, transparent, jelly-like, sessile formations, and consist of amyloid degenerated tissue.

Symptoms depend on the size and seat of the particular tumour. They may be absent or insignificant if the tumour grows slowly or arises at a place which is functionally of no great importance, *i. e.*, on the epiglottis or aryepiglottic fold. Otherwise, they cause disturbances of the voice, respiration, sensibility, and swallowing, or cause paroxysms of coughing.

(*a*) *Disturbances of the voice* are the most common of all the symptoms; for the vocal cords form a seat of predilection for new-growths. Hoarseness is sometimes the only sign pointing to a tumour of the larynx. The alteration of the voice is the greater the more the adduction or vibration of the vocal cords is impeded. Larger-sized tumours on the margin of the vocal cord cause excessive hoarseness, or even aphonia. On the other hand, the voice need not be altered by small excrescences arising from the edge of the vocal cords, especially if they are soft; at least, not during speaking, though perhaps the voice, as a consequence, soon becomes tired. Alteration of the voice, however, is conspicuous during singing, more especially when singing *piano* and *decrescendo*. For the *forte*, the blast of air is usually sufficient to make the vocal cords tense, and to approximate them closely in spite of the slight hindrance. If, however, such a small tumour divides the glottis into two sections, then the voice sounds *doubled* (diphthonia, see p. 387), and easily passes into *falsetto*. If a pedunculated tumour, such as a polypus, is at one time caught between the glottis and at another time escapes from it, the voice sounds now hoarse, then more or less clear and pure. Lastly, tumours lying on the upper surface of the vocal cords may arise from the ventricular folds, ventricle, or epiglottis, and may have an effect like a damper, *i. e.*, a muffling effect.

(*b*) *Disturbances of respiration* almost always exist if large tumours or masses of tumours, such as multiple papillomata in children, protrude into the laryngeal lumen; or the hindrance to respiration may be only transient if a large papilloma is, for instance, temporarily caught between the glottis. Polypi arising above the vocal cords are sometimes caught during inspiration, while subglottic tumours are driven into the glottis by the current of expiration. In the former case the glottis may be once more freed by expiration; and in the latter case, by the inspiration, so that this change of respiration, whereby it becomes alternately obstructed and freed by expiration and inspiration regularly, is of great diagnostic value. Occasionally the "catch" might, however, cause alarming symptoms of suffocation. Bodily efforts and emotional excitement also are liable to increase the dyspnoea, and, lastly, a sudden change of position, *e. g.*, from the recumbent into the upright, might be followed by a dangerous dyspnoea, because of sudden momentary closure of the glottis.

(*c*) *Disturbances of sensibility*, if present, manifest themselves as all kinds of paraesthesiae, such as the sensation of tension, pressure, or of a foreign body being present, etc. *Benign tumours do not usually cause pain.*

(*d*) *Dysphagia* (hindrance to swallowing) is rare.

(*e*) *Cough* is a prominent symptom if the tumour is associated with laryngitis, and especially in pedunculated tumours, which are likely sometimes to cause violent coughing paroxysms or spasms by touching sensitive parts of the mucous membrane. Patients often complain of irritation in the throat.

Diagnosis.—As the clinical symptoms are so little characteristic, the proof that there is a tumour in the larynx can only be provided by the laryngoscope. Larger sized or multiple tumours are not likely to be overlooked. Small tumours, however, often escape discovery, especially if they are hidden in some fold or corner, and do not contrast in colour with their surroundings. Examination during phonation and respiration is indispensable, for many tumours appear different according to whether the glottis be opened or shut. We may by this, at least, arrive at some idea of their consistency, and in not a few cases the tumours can only be recognised by the different appearances they present during phonation and respiration with regard to size and outline, owing to the different shades of illumination during both actions. New-growths in the region of the anterior commissure very easily remain undiscovered, or cannot be seen completely.

In young children diagnosis is always difficult. In the General Section (see p. 368) it has already been shown how we should deal with such cases. Anyhow, we should bear in mind that persistent hoarseness in children is most commonly caused by small nodules on the vocal cords (before described) or by papillomata; rarely, however, by congenital membranes between the anterior ends of the vocal cords. (See p. 406.) If dyspnoea is also present besides hoarseness, then the probability of its being due to papilloma becomes now almost a certainty.

If a tumour has been discovered by the mirror, its seat, size, shape, and consistency must be ultimately ascertained by means of a probe, after previous local anaesthetisation.

We must further note, firstly, whether there is a true new-growth or whether the tumour which presents itself is merely a defined or circumscribed thickening, or a circumscribed inflammatory product of a catarrh or other dyscrasic feature; secondly, whether the tumour is malignant or not; and thirdly, what is the type of the tumour.

1. *Whether we have to deal with a tumour or not* is in many

instances easily answered; yet sometimes it is not always so easy. It goes without saying that certain pathological formations, such as "singer's nodes," prolapse of the ventricle, etc., are not true tumours in an anatomical sense, but, as has been pointed out, are only inflammatory products that are classed among tumours because of their clinical appearances. More important, however, than this distinction is it to discriminate between true new-growths and those tumour-like granulations (sometimes well defined) of a tuberculous, syphilitic, or catarrhal nature. It is well to remember, however, that these latter hardly ever occur in the larynx exclusively, and chiefly occupy the posterior wall and arytenoid cartilages, etc.; which are just the sites not chosen by new-growths. In special cases a microscopic examination must be resorted to, or a trial with iodide of potassium, and a thorough examination of the whole body, and close inquiry into the previous history will be needed.

2. *Whether a tumour is malignant or not* can only be correctly answered by the microscope, provided that the excised portion has been taken from a sufficient depth; otherwise errors cannot be avoided. Benign tumours are excluded if infiltration or inflammation can be noticed in the adjacent tissues; if they appear broken down; if pain and dysphagia are prominent; and if the tumour is growing rapidly.

3. *The type of the tumour* may be recognised by the mirror. Multiple tumours, which have the appearance of condylomata, or cauliflower, or raspberries, are not easily mistaken, nor are the larger sized cysts, or "singer's nodes." In other cases the microscope will clear up any doubts.

It is surely no calamity for the patient if a solitary papilloma has been mistaken for a fibroma; in either case the tumour must be excised. What is practically of the greatest importance only is the distinction between benign and malignant tumours and dyscrasic fungations. *Hence one should never neglect to microscopically investigate every case of intralaryngeal tumour.*

Prognosis.—The prognosis is determined by the type of the tumour. In benign tumours it is favourable as regards life and ultimate cure. Even papillomata, though they sometimes grow quickly and show a distinct disposition to recurrence, do not alter their benign character. But they may also, as has been pointed out, occasion danger from suffocation in small children, in whom the treatment is often beset with many difficulties, apart from the discomfort caused by a preventive tracheotomy.

The smaller the tumour and the easier it can be removed, the better is the prognosis. The contention of *Lennox Browne*, that the operative manipulations act as stimuli to cause a benign tumour to change its character, may be considered as disproved by *Semon*.

We must, however, make a reservation in prognosis with regard to cure in those patients who, for professional reasons, need to take great pains to preserve the purity and capacity of their voice. Although we may often have good results, it will be well not to promise the patient too much.

Treatment.—Some cases of inflammatory, tumour-like formation of a slight degree, as, for instance, "singer's nodes," may be cured if they are cauterised with caustic points of silver nitrate, and the voice is kept absolutely silent. Children's nodules, however, also disappear *in spite of treatment*. Tracheotomy, which is performed on account of threatening dyspnoea, has the effect of causing papillomata to disappear by the functional rest it affords. Setting aside cases in which a pedunculated papilloma has been torn off and brought up by forcible coughing, we may say that benign tumours must be removed by operation, viz., *by intralaryngeal operation*, and only in exceptional cases by extralaryngeal methods.

Intralaryngeal Operations.—Owing to the great variety of the cases, no hard-and-fast rule can be given as to the use of instruments, and it must be left to the discretion of the operator as to which way he will elect to proceed, for in some cases changing the instrument during operation is unavoidable. In any case it is well to test, by means of a probe introduced into the larynx, how far the instrument used should be bent. The patient must be trained to tolerate the insertion of instruments; and as regards this and local anaesthesia, we refer the reader to the General Section of this Part (see p. 399, et seq.).

Stalked or cone-shaped tumours may be removed by the snare or guillotine. The loop of the snare should be just so large that it can slip over the tumour. One must be careful to really cut through the base of the tumour without any pulling; for nothing could be more inconvenient than to have to withdraw the wire from an already half-cut-through growth, on account of some untoward incident, such as the sudden onset of coughing, etc. In other cases, cutting instruments may be used, viz., a pair of sharp cutting forceps or a guarded knife. The laryngeal knife has this advantage in that it permits delicacy of manipulation, especially where the field of operation is very small. On the other hand, it has the disadvantage that a tumour clean cut through easily falls down into the trachea, and a tumour partially cut through has still to be removed by forceps or snare. Given the same conditions, the cutting forceps, or rather the scooped forceps or curette, is, therefore, preferable. The instrument must be inserted with the jaws closed, and then these are opened

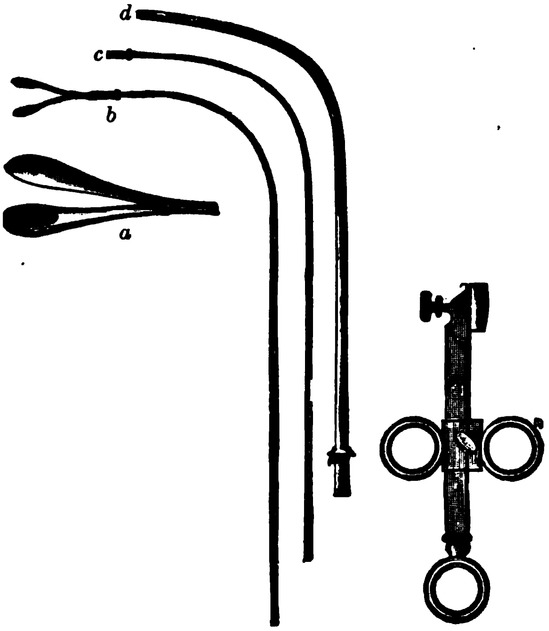

Fig. 195.—Revolving scooped forceps, adaptable to *Krause's* universal handle: *a*, Sharp scooped forceps; *b*, directing staff fitted with the forceps; *c*, directing staff without forceps; *d*, guiding tube; *e*, handle. (The forceps are especially adapted for small tumours which arise from the surface of the vocal cords.)

after the instrument has been brought into the exact position needed; and so the whole tumour, if possible, is then seized and cut away. Tumours of larger size require repeated attacks, and must be removed step by step. In any case great care must be taken while cutting not to drag at the instrument, in order to avoid injury of the vocal cords; and, on the other hand, one should make it a matter of routine to remove the matrix when removing a readily recurrent papilloma. If one does not succeed in removing the matrix, at least it must be afterwards cauterised with caustic points or trichloracetic or chromic acid.

Small cysts may be removed in toto by means of forceps, and large ones may be punctured, or a piece of the cyst-wall may be excised and afterwards iodine or trichloracetic acid may be brushed over the cyst.

Galvano-caustic instruments should be avoided, on account of the violent reaction they produce; and only in exceptional cases may they be used, viz., the red-hot wire for polypi with hard pedicles, or in tumours of a hard and very vascular nature. The loop of wire must first be constricted as much as possible, and then made hot, cutting step by step at intervals. Small angiomata can be destroyed by means of a pointed burner.

Special difficulties are met with in operations in the region of the anterior commissure, and in the cases of small children. In the former case it is sometimes only possible to operate during phonation, or in such a way that the epiglottis is lifted by means of an elevator.*

In children, in whom the papilloma is almost always multiple, very often an operation can only be performed by making use of *Kirstein's* or *Killian's* method of autoscopy; and the operation must be frequently repeated until the matrix is eradicated. Sometimes it is only after years have elapsed that we see our trouble rewarded by entire success. *In threatening dyspnoea any operation must be preceded by tracheotomy.*

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*Translating editor's foot-note: After cocainisation, the epiglottis may be transfixed and drawn out of the way by means of a silk thread passed with a pair of *Kurtz's* suture forceps, suitably modified. This thread can be held by the patient, and so leaves both the operator's hands free for his work. No evil result follows this exceedingly simple procedure—"bridling the epiglottis."—F. R.

Voltolini's method of "sponging," which aims at rubbing off and afterwards wiping away soft tumours by means of a kind of sponge fixed on a holder, is *dangerous* and therefore *objectionable.*

Complications.—Among these may be mentioned: leaving the tumour behind in the larynx, or allowing it to fall down into the lower air-passages; lastly, severe haemorrhage.

The tumour is usually expectorated at once or a little later; large tumours, however, may cause severe dyspnoea if they have escaped into the trachea or farther down. Severe haemorrhage or after-bleeding has been occasionally observed; if this occurs and is persistent, then laryngotomy is required.

After-treatment.—*Absolute silence and rest* must be insisted on. This the more so on account of the reaction following the operation, which not infrequently increases the disturbance of voice which may have already existed. In any case it would be wise to tell the patient beforehand of this latter circumstance.

Extralaryngeal Operations.—The removal from within of a benign laryngeal or tracheal tumour may be contraindicated in spite of surgical skill on the part of the operator, under the following circumstances: 1. If the patient is hypersensitive and, therefore, local anaesthesia is not of much service. 2. If the tumour is very large, hard, and vascular. 3. If access is in no wise gained per vias naturales. These suppositions, singly or conjointly, are not frequently encountered. But if the patient asks for removal of the tumour in spite of those drawbacks, or if there are cogent reasons for operating, then and only in these circumstances must the larynx be opened from the outside. One of the following methods might be employed: (*a*) Subhyoid pharyngotomy, *i. e.*, the division of the ligamentum hyo-thyroideum below the hyoid bone; (*b*) infra-thyroid laryngotomy, *i. e.*, division of the crico-thyroid (conical) ligament. (*c*) Thyrotomy—division of the thyroid cartilage.

Subhyoid pharyngotomy is indicated only in large tumours of the epiglottis; *laryngotomy*, in subglottic new-growths. *Thyrotomy*, which has up to the present been resorted to chiefly in exuberantly growing papillomata, affords convenient access, but it suffers from a great disadvantage, viz., that the divided thyroid cartilage does not afterwards join together very symmetrically in spite of the greatest care, so that it might happen that one vocal cord may stand higher than its fellow, and very marked hoarseness, and even aphonia, may consequently result. Such a risk is not worth running in operations on benign tumours, apart from the fact, also, that thyrotomy does not prevent recurrence of the papillomata. This risk, however, can be partially avoided if only the *lower part of the thyroid cartilage is divided,* and if *the upper part,* with the attachment of the vocal cords, *is left untouched.* But even this form of partial thyrotomy, as well as the other methods of extralaryngeal operations, are of a much more serious nature than the intralaryngeal operations. In cases of benign new-growths, therefore, they are only permissible in special and exceptional circumstances.

B. MALIGNANT TUMOURS.

While benign tumours show a great variety in regard to their histological structure, malignant tumours are chiefly met with in two forms, *i. e.,* sarcoma and carcinoma. The former is much rarer than the latter, in the ratio of 100 carcinomata to 4 or 5 sarcomata.

SARCOMA.

Pathology. (See Figs. 196, 197.)—Laryngeal sarcoma occurs as a primary or secondary affection. In the latter case it is metastatic, or, what is oftener the case, it consists of a direct invasion by the primary tumour growing from neighbouring organs and tissues.

In one case under my observation the origin of the sarcoma could be traced to the cervical glands.

Sarcoma shows itself as a well-defined, rounded, smooth or sometimes lobated tumour, of pale or dusky (sometimes greyish white or yellow) colour; its consistency is at times hard, at others softer. The surface usually shows great vascularisation. As a rule, the tumour is sessile, but there have been cases where the sarcoma distinctly showed a pedicle. The new-growth incessantly advances in all directions. If it spreads more superficially, it presents itself as an infiltration, which, however, is not like a tuberculous infiltration, nor, like carcinoma, does it merge into the surrounding tissue, but only penetrates it, but at the same time remains sharply distinct from the healthy tissue. Ulceration is not frequent. The vocal cords are the seat of predilection, but sarcoma may also be found on other parts of the larynx. In the trachea the upper part is usually the seat of the new-growth, which is here mostly secondary, the primary tumour being usually in the thyroid gland.

Histologically, it shows spindle or round cells (spindle-celled or round-celled sarcoma), but occasionally mixed forms are met with, *e. g.*, fibro-, myxo-, chondro-, angio-, adeno-, and melano-sarcoma.

Symptoms.—The clinical symptoms are not much different from those of benign neoplasms. This is chiefly due to the small inclination of the sarcoma to break down. At first disturbances of the voice prevail; later dyspnoea, to which difficulties in swallowing may subsequently be added. Altogether, the patient complains less than in carcinoma of the larynx. The objective symptoms depend on the size and site of the tumour.

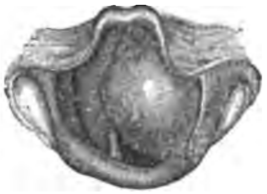

Fig. 196.—Sessile sarcoma of the left ventricular fold (from the Vienna Laryngological Clinic).

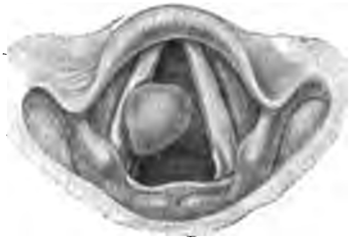

Fig. 197.—Pedunculated sarcoma of the right vocal cord (*Türck*).

Diagnosis.—Confusion is possible with syphilitic and tuberculous infiltration, and with carcinoma or even benign tumours. The more or less distinct limitation, however, from the adjacent tissue; the incessant growth without ulceration tends decidedly to preclude tuberculosis, syphilis, and carcinoma. The cervical glands usually show no enlargement, though this might occur. A positive reaction to iodide of potassium does not exclude sarcoma. In the case above mentioned, which was under my observation, the tumour distinctly diminished under the influence of large doses of iodide of potassium, and yet turned out to be a true round-celled sarcoma of the right vocal cord. Diagnosis can only be *assured with certainty after microscopic examination of an excised portion.*

Prognosis is not as bad as in carcinoma; comparatively, it is best in spindle-celled sarcoma, which grows, for the most part, slowly. The soft tumours, however, spread rapidly, infiltrate the lymphatic glands, form multiple metastases, and show a certain inclination to ulcerate. Death ensues with symptoms of exhaustion, suffocation, septic pneumonia, or metastases in other organs.

Treatment is identical with that for carcinoma, and will be discussed under that heading. Inoperable cases may be treated with arsenic. The chances of treatment will perhaps improve under the influence of radium and *Röntgen* rays. Occasionally intralaryngeal or extralaryngeal operations have proved successful, in some cases, at least, for just as long as they were kept under observation.

2. CARCINOMA.

Cancer of the larynx, like every other cancer, is a tumour derived from preëxisting epithelial cells, and is of epithelial or epithelioid character. It arises in a part which is not usually occupied by epithelium, and whence its malignancy is supposed to originate. This heterotopia is, as *Orth* expresses it, a morphological landmark of destructibility and also of malignancy. It is chiefly from the squamous epithelium of the larynx that laryngeal carcinoma takes its origin, and for this reason the majority of cases belong to the squamous epithelial type of carcinomata.

Cylindrical (cubic) celled and *glandular* celled carcinoma are rare in the larynx, but it occasionally happens that on places which are normally covered with cubical epithelium a squamous celled carcinoma developes, by some process of metaplasia of the epithelium. The tumours arising from *glandular epithelium* show a very scanty stroma of connective tissue, and represent the *medullary* (soft) and very *malignant form of cancer.*

In a few cases, however, the fibrous stroma is so abundant that the carcinoma assumes the form of *scirrhus* (*fibrous carcinoma*).

Primary Cancer.—Cancer of the larynx is almost always primary, and chooses as its site of predilection the vocal cord (covered with squamous epithelium). Next to these come the ventricular folds, next, the epiglottis, and finally the pharyngeal surface of the posterior laryngeal wall. Other parts of the larynx are rarely the primary seat of the carcinoma. Sometimes, but not frequently, the carcinoma spreads into the larynx from the neighbourhood; and, furthermore, carcinomatous metastases in the larynx are very rare.

In the larynx, as elsewhere, carcinoma exhibits the well-known disposition to spread into adjacent tissues, by sending forth well-recognised epithelial columns, and forming epithelial cell-nests. The developmental energy is not at first very great, and it usually takes two or three years or longer before a carcinoma of the vocal cords is sufficiently far advanced to cause marked disturbances. Later on, however, it changes its slow nature of growth and becomes very aggressive. In certain kinds of cancer, more especially in the so-called soft forms, rapid development is very pronounced. The quicker or more rapidly a carcinoma developes, the sooner does it begin to ulcerate, on account of its scanty blood-supply. The ulceration spreads superficially and in depth, and then putrefaction and decomposition soon supervene. The progressive ulceration and incessant spreading of carcinoma into the adjacent parts involves or infiltrates more and more of the tissues; for it does not stop short at cartilage, but invades the latter also, giving rise to perichondritis and necrosis of the cartilage. Having destroyed the latter, it continues its course to the neighbouring organs. There is no halt, except that called by death.

Two things are noteworthy: first, that the entire process may become arrested for a certain time for some unknown reason, only to advance more rapidly afterwards; secondly, that the lymphatic glands are not frequently infiltrated *until late in the disease*, and that metastases also occur somewhat late. With regard to the latter point, there is a difference between the so-called *internal* and *external* laryngeal cancers. To the former, according to *Krishaber*, belong those which arise from within the larynx in the region of the vocal cords, and to the latter belong those cancers arising in the vicinity of the larynx, that is, from the epiglottis, arytenoid folds, pyriform sinus, and pharyngeal surface of the posterior laryngeal wall.

Internal laryngeal cancer infiltrates the lymphatic glands less quickly because of the scanty supply of lymph-vessels in the region of the glottis, and it may, therefore, claim the credit of being a purely local process for a longer time than the very dangerous *external laryngeal carcinoma.* The glands at the anterior border of the sterno-cleido-mastoid muscle (glandulae concatenatae) are the first, and often the only, glands infiltrated, but the submaxillary glands are also occasionally affected.

Secondary carcinoma usually occurs at first at a site which is generally and obviously concerned with a primary focus; *e. g.*, tongue, pharynx, gullet, thyroid gland, etc. As the infiltration of the adjacent tissue presupposes an already very advanced stage of the primary growth, the larynx, in such a case, is usually and already so much involved that it is often most difficult to say which was the primary and which the secondary tumour.

Symptoms and Course.—*Two stages* can be distinguished in the clinical course of laryngeal carcinoma: One, where the growth still shows, seemingly, at least, a local character; and a second stage, where other organs have already been involved and the whole organism has, so to speak, become infected.

I. *Stage of Local Growth.*—This takes from one to three years; and with regard to diagnosis and treatment, this is most important for the medical attendant. As long as the cancer is localised or confined to the larynx, there is a possibility of effecting a cure, for a certain time at least, by destroying the focus of the disease. All depends, therefore, on an early and correct diagnosis. Unfortunately, an early diagnosis from the clinical symptoms is not always easy. It is just laryngeal cancer which, in the later stages, shows such appalling powers of destructibility. We here particularly refer to primary carcinoma, which begins, as a rule, with insignificant functional disturbances. There is merely a slight hoarseness complained of, which is by no means characteristic, but very persistent. Hoarseness occurring in persons of middle or older age, without other signs of laryngeal disease, should always render us suspicious. Hoarseness may, however, be missing if the carcinoma arises from somewhere in the vicinity of the larynx (see above); but here, however, difficulties in swallowing (dysphagia) are rather more prevalent. The general health at this stage is usually undisturbed.

Gradually, the hoarseness increases; patients then complain of pain on swallowing, speaking, and coughing, and the pain also occurs spontaneously, particularly at night, and radiates towards the ear. The dysphagia may be due to the actual presence of the tumour or to ulceration. The larynx becomes narrowed, and dyspnoea ensues: feeding is rendered more and more difficult, and although the general health might now suffer thereby, yet the disease still remains localised.

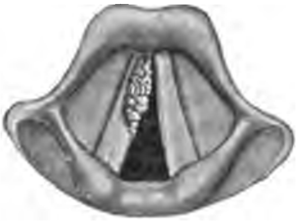

Fig. 198.—Small tuberous carcinoma of right vocal cord (*Jurasz*).

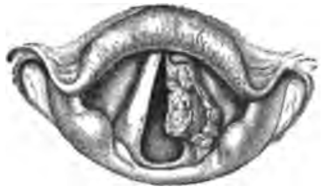

Fig. 199.—Carcinoma covering the entire left vocal cord and showing ulceration (*Türck*).

The laryngoscopic examination will show different pictures as to site and size and extent, which latter depends much on whether the surface of the tumour is already ulcerated or not. (See Figs. 198, 199, 200, 201, 202, 203.)

The *surface being intact*, the carcinoma appears in *two forms:* either as a well-defined tumour or as a diffuse infiltration.

On the vocal cords the defined type of tumour is usually found as a flat prominence with smooth, but oftener tuberous, surface (*Fränkel's* carcinoma polypoides). Its colour varies from white to red. Signs of inflammation in the neighbourhood may be present or missing, and in the former case one gets the impression that the tumour is not merely resting on the vocal cord, as, for instance, a fibroma appears to do, but that it really appears to *grow out* of the vocal cord.

Diffuse infiltration is rarer than the polypoid form, and the vocal cord then appears diffusely thickened. At first, such an infiltration exactly resembles a tuberculous or syphilitic infiltration, and even a pachydermia (see p. 455) may show the same picture. Later, the carcinoma assumes a more tuberous and granulated appearance, but is still very much akin to pachydermia.

Diminished mobility of the vocal cord, which is considered by *F. Semon* as pathognomonic of carcinomatous infiltration, is certainly often missing; but it is always present if the carcinoma is seated on the posterior third of the vocal cord. The reduced mobility is, however, not characteristic of carcinoma alone, for all pathological changes in the region of the crico-arytenoid joint are accompanied by reduced mobility of the vocal cords.

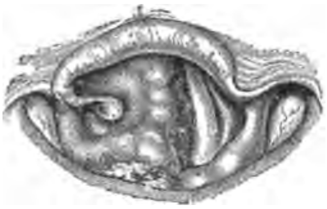

Fig. 200.—Carcinoma of the left side of larynx, showing ulceration at the posterior wall, spreading on to the epiglottis (*Türck*).

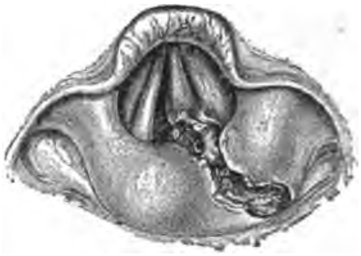

Fig. 201.—Carcinoma of left side of larynx, showing ulceration, perichondritis, and oedema (*Türck*).

The polypoid and diffuse forms of carcinoma are also found on other parts of the larynx, but there the colour is mostly of a bright red, and the surface soon becomes granulated or tuberous. Carcinoma, which arises primarily from the walls of *Morgagni's* ventricle (*carcinoma ventriculare—Fränkel*), is at first invisible, and merely pushes the aryepiglottic and ventricular folds forwards. Both folds thus appear reddened and later on uneven or tuberous also; and the vocal cord may be obscured or hidden by it. Carcinoma *developing in the deeper tissues* (*M. Schmidt*) may also remain unrecognisable for a long time, and then only manifest itself by the bulging of the overlying tissues and by the formation of papillomata (see p. 528), which must then be regarded as inflammatory products due to the irritation set up by the carcinoma. If one finds multiple papillomata always recurring after a repeated operation in an adult, then such a person should be suspected of having carcinoma.

If we see the patient later, the picture will have totally changed. The mucous membrane then appears lumpy and nodular, and the tumour has a fissured, cauliflower-like appearance.

As the tumour penetrates in depth, this fact is manifested by diminution of mobility, by reddening, swelling, oedema, and perichondritis, etc., and superficial extension may be recognised by the fact that several other parts of the larynx are already affected. The tumour may have already infiltrated the whole of its own side and have also extended across to the other side.

At the commencement, ulceration is rare, but the softer the carcinoma and the more the affected part is exposed to irritation, as from swallowing, then the sooner does ulceration show itself. The carcinomatous ulcer somewhat resembles a gummatous ulcer, as both of them possess thickened and everted edges. On the floor of a carcinomatous ulcer, however, newly formed carcinomatous nodes may frequently be seen.

II. *Stage of Complications and General Infection.*—This offers no difficulties to diagnosis, but insurmountable obstacles, however, to treatment. The patient's sufferings are already very much aggravated by the incessant growth of the tumor. Pain on swallowing is increased, and renders feeding exceedingly difficult; dyspnoea may require tracheotomy, and an offensive foetor, due to the decomposition of the tumour, is not only perceptible to the patient himself, but is also very unpleasant to his friends. Hoarseness soon becomes complete aphonia, even at an already early stage, without a tracheotomy. The patients often "swallow the wrong way," and are racked by severe paroxysms of coughing.

The picture is still more complicated if other organs are affected. The lymphatic glands become swollen in *external* laryngeal carcinoma earlier than in *internal* (intrinsic) laryngeal cancer (see p. 542), and this circumstance signals the beginning of the end. Laryngeal cancer once having trespassed beyond the larynx or having infiltrated the cervical glands, *is no longer a local disease,* and it is only a question of time when the exhausted patient will succumb to suffocation or exhaustion. Suffocation usually ensues from increasing stenosis, or if tracheotomy had been previously performed, from obstruction of the tracheal canula. On the whole, cachexia appears rather late, owing to the slight predisposition of laryngeal carcinoma, especially at the beginning, to form metastases. An intercurrent pneumonia or a severe haemorrhage frequently puts an end to a painful existence.

In the second stage the mirror shows us a picture which could hardly be misinterpreted. Above all, the association of tumour with ulceration is characteristic, and the extension into adjacent tissues conspicuous.

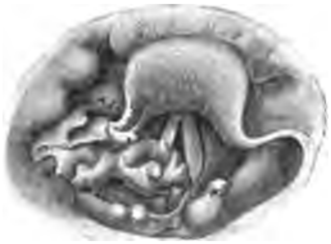

Fig. 202.—Carcinoma of right half of larynx, spreading to the pharynx and tongue (*Türck*).

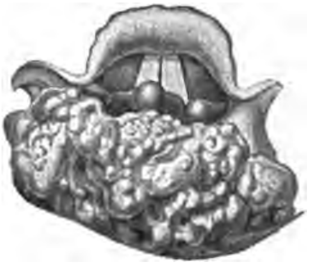

Fig. 203.—Carcinoma of lower pharynx (hypopharynx), spreading in front into the larynx. Secondary carcinoma of larynx (*Türck*).

The region of the larynx bulges, and the tumour may be felt from the outside or has already perforated the skin.

Diagnosis.—In the advanced stage the clinical features, together with the laryngoscopic appearances, will leave no doubt as to the nature of the process. But in its early stages, and especially at the commencement, the diagnosis is frequently very difficult, as it is quite easy to mistake carcinoma for tuberculous, syphilitic, or catarrhal pachydermic infiltration, or even for a benign new-growth.

It can easily be understood that one may have doubts concerning a carcinoma which may be growing in the deeper structures, or in a case of ventricular cancer; and only, indeed, if one is able to notice small unevennesses of the infiltration are one's suspicions then aroused. On the vocal cords, every circumscribed redness, swelling, or prominence, or, as one should almost say, every circumscribed abnormality, if it occurs on one side only, or on the posterior part of the vocal cord in *elderly people*, should awaken our suspicions, the more so if it is accompanied by hoarseness. If one finds the mucous membrane reddened at several places which are separated from each other by areas of healthy appearance, then carcinoma may be excluded. Pachydermia mostly occurs bilaterally, and preferably on the region of the vocal processes. One-sided pachydermia, on the other hand, as well as circumscribed papilloma, may mask a developing carcinoma. To make a diagnosis of carcinoma from an ulcer alone is, for the most part, impossible.

The difficulties enumerated in the foregoing, and which are met with in the diagnosis of early cancer, compel us to resort to other means besides the laryngoscopic examination, viz., anamnesis, examination of the lung, and, above all, however, to a probationary *administration of iodide of potassium and a microscopic investigation of a piece excised from the deeper part of the growth by means of a double curette* (cutting forceps).

We have already pointed out how important is the microscopic examination of all tissue that is removed from the larynx. In such cases one who controls his investigations by the microscope will protect himself and the patient from unpleasant surprises. Nevertheless, the great advantage of microscopic investigation must not be overrated. *Only a positive observation* is a test. A negative result is of no value because one may have doubts as to whether one has really examined a portion of the tumour proper. One should, therefore, take care that one does not excise too small a piece, and that it is also taken from the deeper parts of the suspected growth—of course, after previous local anaesthesia. If the result of the examination is negative, then the excision from an adjacent spot must be repeated. The excised portion must be hardened, stained, and cut perpendicularly to the surface, as otherwise one may mistake the papillary tissue or the superficial epithelium for carcinomatous epithelial processes (columns) (see p. 454). The value of microscopic examination may also be diminished if a mixed disease is present, as *sometimes carcinoma is complicated by syphilis or tuberculosis.*

If a probationary excision is impossible for some particular reason, or the examination was negative, then we must leave the diagnosis in suspense and await the further course, but meanwhile, however, iodide of potassium must be given in large doses. Tertiary syphilis and laryngeal carcinoma have been so often confounded that one should not hesitate for a moment to appeal to iodide of potassium in any doubtful case. If large doses are ordered, then in about two weeks one will probably know how the matter really stands.

Prognosis.—*Carcinoma, when recognised early, is curable.* A lasting cure, however, may only be expected if the carcinoma can be totally eradicated; otherwise recurrence is unavoidable. Tracheotomy, if performed in time, is likely to prolong life for months and even for one and a half years; on the other hand, it may furnish an opportunity to the neoplasm for further extension.

On an average, the duration of laryngeal cancer is from about one to about three years, but if it does not grow rapidly and if it arises from the deeper tissues, then even four or five years may elapse. Frequently, especially in the first stage, a shorter or longer interval of arrest of growth may take place.

Treatment.—Considering that only a complete and thorough eradication of the tumour, cutting far into the healthy tissues, can be of any lasting effect, and owing to the fact that the tumour usually extends further than can be seen by the mirror, one will easily understand that only an *extralaryngeal operation* will be of any value. The possibility of establishing a permanent cure by an intralaryngeal operation has been proved by various authors, and cases are on record where cure has been established, at least for a considerable period. These, of course, can only be those cases where one has an opportunity of seeing by the mirror a clearly defined initial infiltration, which can be radically removed, and the patient kept under observation, so that a thyrotomy (laryngofissure) might be at once performed if a recurrence should necessitate it. But in the case also of an *intralaryngeal operation*, this must be done as thoroughly and as radically as possible, and this circumstance renders the intralaryngeal operation very much the same as a laryngofissure, considered, at least, from the surgical point of view, which, thanks to the enormous improvement in modern operative technic, need not be ranked as a serious operation. Recently *v. Bruns* has collected 117 cases of laryngofissure, reaching as far back as 1890. Of these, 48 per cent. were cured for one year, 22 per cent. showed recurrence, and 9 per cent. died. These figures, however, do not give a true account of the good results achieved by laryngofissure, for they are much influenced by the incredibly bad results of some operators. *Semon* had only one death in twenty operations, two recurrences, and seventeen cures of from one to three years' duration. *B. Fränkel* opposes to these figures his own 32 cases of intralaryngeal operation, of which 12.5 per cent. were permanently cured, *i. e.*, for at least three years, 12.5 per cent. remained free from recurrence for one year, and 40.7 per cent. recurred within one year. This latter list is not statistically of much importance. The fact that an intralaryngeal operation is a great incitement for recurrence from carcinomatous tissue accidentally left behind after operation, and only causes it to fungate and ulcerate the quicker, is a matter for serious consideration, and is not seldom the reason for missing the psychical moment for performing a radical operation.

It is necessary, also, to watch the patient after a laryngofissure, but he himself will be less anxious as regards his mode of living in the future, knowing that a "radical" operation has been done. Considering how much the public already knows about medical matters, and how wide-spread is the fear of cancer, the patient will probably not allow himself to be deceived about the seriousness of his disease. Though it is not necessary to tell him all about the nature of his disease, we are bound to explain to him the gravity of the situation and must advise him to undergo laryngofissure. If he still refuses the extralaryngeal operation, then, and only then, may we satisfy him and ourselves by doing an intralaryngeal operation.

Following up these premises, we come to the following conclusions as regards the indications for certain operative methods:

(1) *An intralaryngeal operation* is indicated in clearly defined initial cases of internal cancer, when extralaryngeal operation has been refused:

(2) *Laryngofissure* (laryngotomy), in all clearly defined initial cases of internal cancer, as long as it is confined to the internal soft parts, *e. g.*, to the vocal cords, and has not spread much.

The operation can be performed under local or general anaesthesia, but it will be mostly necessary to perform tracheotomy some time or days previous to the operation, and to insert a *Trendelenburg's* tampon-canula, in order to prevent the blood running down into the trachea during operation. Instead of resorting to tracheotomy and a tampon-canula, one may operate with the head hanging at a lower level than the shoulders, on an inclined plane (*Rose's* position). The skin is incised exactly in the middle line, immediately below the hyoid bone, and the incision is carried down as far as the cricoid cartilage, dividing the skin, subcutaneous tissue, and fascia; the muscles are detached with a blunt dissector and pushed out of the way, and all bleeding vessels are secured. The ligamentum conicum is now punctured by means of a pointed knife, and the thyroid cartilage is divided upwards from this opening by means of scissors or bone forceps (partial laryngofissure, thyrotomy). In dividing the thyroid cartilage, the median line must be rigidly adhered to, particularly in order to conserve the attachment of the vocal cords. The larynx being now opened, the mucous membrane is then painted with cocaine or adrenalin, in order to avoid unpleasant coughing and bleeding. For the illumination of the larynx, an electric head-lamp may be used. The tumour is now circumcised down to the perichondrium, and *removed close to the cartilage.* If one finds, after opening the larynx, that the tumour is larger than was expected, or that the partial laryngofissure does not afford full access to the tumour, then the cricoid cartilage may be divided as far down as the trachea (total laryngofissure). The surface of the wound must afterwards be cauterised with the galvanocautery, and iodoform then rubbed in. After the operation, neither tamponading nor a canula is necessary, as their absence tends to simplify matters. The thyroid plates are united by their perichondrium and also the upper end of the cutaneous wound, while the lower end of the wound in the region of the trachea remains open and is covered with iodoform gauze. The after-treatment is simple, and the patients are allowed to swallow from the first day if they are able to. The wound usually heals in two weeks. (For further details see text-books on surgery.)

The functional result after laryngofissure is usually good. A vocal cord which may have been necessarily removed is usually replaced by a fibrous band, which assumes the rôle of the vocal cord. In any case the patient should be warned of the disturbance to the voice which may be expected. One should never allow the phonetic function to exercise an influence upon the operation, however, and should always operate through healthy tissues.

(3) *Subhyoid pharyngotomy* in cases of *external laryngeal carcinoma*, where the tumour is confined to the epiglottis and aryepiglottic fold.

A preliminary tracheotomy is first performed, and is followed by the insertion of *Trendelenburg's* tampon-canula; an incision is then made close below and parallel to the lower margin of the hyoid bone, about 7 cm. in length across the neck, dividing the skin, superficial fascia, and muscles; the ligamentum hyothyroideum is then seized by two pairs of forceps and divided; next, the thin covering of the valleculae is incised, and the incision prolonged outwards at both ends. The larynx can now be lifted somewhat through the wound, and any tumour situated in the neighbourhood of the epiglottis and aryepiglottic fold can be circumcised freely into the deeper and more healthy tissues. All bleeding vessels must be secured, and in order to prevent pneumonia from aspiration of blood, etc., the wound must be plugged very carefully. *Gluck* endeavours to establish a provisional plastic occlusion of the larynx by stitching the tampon together with the surfaces.

Unfortunately, the results of subhyoid pharyngotomy are occasionally upset by the occurrence of pneumonia from aspiration of discharges, and by the infiltration of the glands, which occurs very early in external laryngeal cancer.

(4) *Partial Extirpation of the Larynx.*—For all cases of internal laryngeal carcinoma which have advanced beyond the mucous membrane and into the cartilage of one side of the larynx. If the tumour is close to the anterior or posterior commissure, we must expect that the epithelial columns will have been already growing into the other side, in which case only total *extirpation* will be justifiable.

(5) *Total Extirpation.*—For all cases of internal laryngeal carcinoma which has spread on both sides of the larynx.

Partial extirpation will frequently become necessary as the second act of a laryngofissure if, after opening the larynx, one finds that the carcinoma has already advanced unexpectedly into the cartilage. The ultimate results of partial or total extirpation are, of course, much worse than laryngofissure. Nevertheless, they have lately become somewhat better than was formerly the case, owing to the improvements in method and the subtle elaboration of plastic operations which have enabled the skilled surgeon to better avoid pneumonia and sepsis caused by the aspiration of blood or discharges. The voice often remains fairly good after *partial extirpation*, and swallowing also may go on without much difficulty. After *total extirpation* the patients must be satisfied with a false voice, which they learn to produce by muscular action at some part of the pharynx, or they must wear an artificial larynx, which, however, is not always very pleasant.

Inoperable cases, of course, can only be treated symptomatically. This can only mean the relief of suffering, and here great patience is required on the part of both surgeon and patient. In threatening dyspnoea tracheotomy will be necessary, as it provides relief for a time and helps to prolong an existence which is only nominally such.

Radium and the *Röntgen* rays will perhaps prove useful in the future, provided that the technic of their application is also improved. Lately, *Mader*, as has been already mentioned (see p. 498), has devised a suitable laryngeal tube which allows the exposure of the epiglottis, posterior wall, and sometimes the interior of the larynx, to the *Röntgen* rays. In inoperable cases actino-therapy is justifiable.

TRACHEAL CARCINOMA.

Tracheal carcinoma is seldom primary, as the cancer usually spreads to the trachea from the larynx, oesophagus, thyroid, or lymphatic glands. Primary carcinoma of soft or medullary structure is usually found in the upper section or close above the tracheal bifurcation. It usually forms a flat, tuberous tumour, which rapidly breaks down and invades adjacent structures.

Symptoms.—The chief symptom, especially in the later stages, is dyspnoea. If the cancer has perforated the gullet, then particles of food may enter the trachea and cause violent coughing paroxysms. Actual cachexia does not occur, because the patients die from suffocation or pneumonia before it developes.

Diagnosis.—Dyspnoea and stridor point to stenosis of the trachea (see p. 413), but direct or indirect tracheoscopy will reveal the cause of the dyspnoea.

Treatment is almost always merely palliative. In threatening suffocation, tracheotomy, superior or inferior, according to the seat and extent of the cancer, must be performed. Sarcoma of the trachea is not quite so unfavourable, because it does not break down and may be more easily operated on.

X. NERVOUS LESIONS.

A. DISORDERS OF SENSIBILITY.

(*a*) *Anaesthesia.*—The *central causes* of anaesthesia are: Hysteria, tabes, bulbar paralysis, and other cerebral focal diseases. Of the *peripheral causes* of anaesthesia, the main one is diphtheria, which often paralyses the motor (inferior laryngeal) as well as the sensory nerves (superior laryngeal) of the larynx, and is frequently also associated with anaesthesia and paralysis of the soft palate. Artificial anaesthesia can be produced by the application of certain drugs, *e. g.*, cocaine, alypin, menthol, etc.

Anaesthesia of the laryngeal mucous membrane can only be tested for by the probe. Touching with the probe does not excite any reflex action. It has been previously pointed out (pp. 304 and 331) that in anaesthesia of the laryngeal mucous membrane, more especially if associated with anaesthesia of the pharynx, mis-swallowing is a common occurrence, which encourages particles of food to enter the lower air-passages, where they are apt to set up pneumonia. Extensive laryngeal anaesthesia is, therefore, always a great danger.

In anaesthesia of the larynx the sense of touch and reflex excitability may be lost, while the sense of pain might be preserved. Such a case is reported by *Schnitzler*, where the probe was not felt, but severe pain was complained of (anaesthesia dolorosa). Diminution of sensibility (hypaesthesia) varies much also in healthy persons within certain limits, so that one might be embarrassed to decide, in a given case, whether pathological hypaesthesia were present or not.

(*b*) *Hyperaesthesia.*—Increased sensibility as well as reduced sensibility sometimes occurs in healthy persons. Some persons react to every stimulation of the mucous membrane with violent cough or even laryngospasm (see p. 397), but the irritability soon subsides, and the persons become accustomed to the stimuli. Sensibility is often abnormally increased by previous catarrhs, particularly in smokers and drinkers, but here the hyperaesthesia is merely a symptom of a local disorder. On the other hand, in hysteria or neurasthenia hypaesthesia manifests itself as a disease *sui generis*, and may be so marked that the patient's mind is quite dominated by it.

Tuberculous or anaemic persons are also very "sensitive in the throat." Comparatively slight irritations, such as a change of temperature, light smoke, or slight pressure on the throat, are sufficient to cause very unpleasant sensations in these persons, *e. g.*, burning, coughing, aching, itching, dryness, etc.; and sometimes the patients are so depressed that they dare not speak other than in a whisper (phonophobia). Not infrequently such a hyperaesthesia is the cause of various reflex disorders, which find their analogues in the nasal reflex lesions previously discussed. (See p. 121.) One should not, however, be too quick to assume hyperaesthesia or reflex neurosis, as most patients are somewhat hypersensitive, especially if they are not gently treated. Very often, moreover, what has been considered to be a neurosis may turn out later, if one examines more carefully, to be an organic disease, viz., a diseased joint or an injury, etc.

If the hyperaesthesia is very considerable, and if it occurs at intervals and is localised to the area supplied by the superior laryngeal nerve, it is spoken of as *laryngeal neuralgia.* The affection is usually unilateral. A pressure point, according to *Avellis,* can be found between hyoid bone and thyroid cartilage, at the spot where the superior laryngeal nerve penetrates the thyro-hyoid membrane. It is the same spot from which the frightful laryngeal crises in locomotor ataxia are elicited. (See p. 582.)

(*c*) *Paraesthesia* may be of nervous, viz., central, origin, or it may be caused by some local disorder, the symptoms of which are manifested in a most peculiar manner. As with paraesthesia of the pharynx, so also laryngeal paraesthesiae are chiefly met with in cases of hysteria and neurasthenia; it very often accompanies anaemia and chlorosis, and is sometimes also a forerunner of pulmonary disease. Foreign bodies, which may or may not have been removed, very often occasion considerable paraesthesia. Hypochondriacs who have once suffered with their larynx and are very assiduous in studying medical books and encyclopedias, preferably localise their abnormal sensations in the larynx, which they consider to be particularly endangered by tuberculosis, cancer, or syphilis. The lay person is, after all, better acquainted with the larynx than with the neighbouring pharynx. That the capacity of localisation, however, is very unreliable has been already pointed out, and one should, therefore, carefully examine any given case, whether the cause of the abnormal sensation be attributed to the throat or larynx. In the throat we can find the exact site of irritation by means of the probe; not infrequently, however, only after a prolonged search (see p. 331). In the larynx reflex excitability is altogether so much more pronounced that we cannot reasonably expect a good result by probing (sounding); besides, the local finding often shows so little difference from the normal that it is not of much assistance.

Treatment should aim, in the first place, at the consideration of the etiological factors, but, as we have seen, this will not always succeed. Local changes must be treated accordingly. The general health must be strengthened by roborants, hardening-cures, bromides, etc.; and, lastly, suggestive treatment must not be neglected. Much that is ordered in hyperaesthesia and paraesthesia acts only by suggestion. We desire to warn against multifarious manipulations, and especially also against prescribing narcotics; though in neuralgia, aspirin, phenacetine, and pyramidon are useful; and morphin in some cases might even be inevitable. Locally, menthol pastilles, or inhalations of menthol, and in very resistant cases painting with silver nitrate (3 to 10 per cent.), may be tried.

For anaesthesia, the galvanic or faradic current may be applied and the patient fed through an oesophageal tube.

B. DISORDERS OF MOBILITY.

I. PARALYSES (HYPOKINESES).

Paralyses and Pareses.—*General Etiology and Pathology.*—The paralyses of the laryngeal muscles resulting in complete or incomplete inhibition of mobility may be divided into: (1) *Myopathic*, where the cause lies in the muscles themselves; (2) *neuropathic*, where the nerve supply is diseased. It can be easily understood that an inflammation affecting the mucous membrane and deeper tissues may also alter the muscular tissues, so that their function becomes disturbed. In this sense the muscular pareses must be understood which occur in the course of catarrhal, infiltrative, or destructive inflammations. (See the chapters on Laryngitis, Tuberculosis, etc.)

Mogiphony, described by *B. Fränkel*, *i. e.*, the condition in which the voice soon becomes tired, and which occurs in professional orators or singers, may be regarded as a myopathic paresis, due to overstrain of the voice or to a too frequent use of unwonted registers. The voice then breaks down and is accompanied by pains in the throat, especially if strained professionally. These "paralytic occupation neuroses" are fostered by abnormal processes in the nose and pharynx which are prone to alter the production of voice and articulation; as, for instance, hypertrophic rhinitis, hypertrophy of the tonsils, lateral pharyngitis, etc. The treatment would have to comprise a complete rest for a long period, as well as the exclusion of noxious agencies.

In most cases of myopathic paralysis the corresponding nerve is probably also affected. We must even assume that the inflammatory process, considering the intimate relation between a muscle and its nerve in the larynx, very easily spreads from the muscular tissue to the supplying nerve branches; and, again, a neuropathic paralysis, if existing for some while, may be accompanied by paresis of the corresponding muscle, through what is called "atrophy of inactivity." Hence it follows that it will very frequently be impossible or at least very difficult to distinguish, by means of the laryngoscopic image, a myopathic from a neuropathic affection. Of pure myopathic nature is the paralysis which is caused by the presence of trichina spiralis. (See p. 520.)

The neuropathic hypokineses extend over a region which reaches from the cortex to the peripheral nerve terminals in the larynx. Accordingly, one distinguishes *central* and *peripheral* paralysis, viz., according to whether the lesion of the nervous apparatus lies in the central nervous system or in the peripheral laryngeal nerve. We are not always enabled to demonstrate the anatomical substratum of a paresis or paralysis, as, for instance, in those functional lesions of hysterical or psychic origin. In order to distinguish functional paralysis from organic lesions, *Semon-Rosenbach's* law will hold good, *i. e., in all functional paralyses of the larynx the constrictors of the glottis (adductors) are almost always affected; in all the organic and progressive organic paralyses, whether central or peripheral in origin, the dilators of the glottis (abductors) are at first or exclusively affected.**

Explanations of this peculiar condition of the two groups of muscles, which are both supplied by the recurrent nerve, are not wanting. *Semon* himself thinks that the cause must probably be sought in some biochemical changes which take place between the various fibers of the recurrent nerve and the muscles (narrowers and dilators) supplied by them. The fibers supplying the dilators form a separate bundle and are supposed to be more sensitive in regard to organic lesions than fibers supplying the constrictors, and, therefore, sooner become disturbed functionally. This clinical observation is corroborated by the experimental fact that the dilator fibers are sooner and more severely injured by thermic, chemical, and electrical stimulation than the fibers for the adductors (*B. Fränkel, Gad, Frese*). There are also exceptions to *Semon-Rosenbach's* rule. *Saundby* reports a case (1903) where the spreading of an oesophageal carcinoma upon both vagi and recurrentes first manifested itself by a paralysis of the adductors, which persisted until death. The abductors prevailed over the adductors until the last moment, and the glottis stood open all the time. But this case is not free from objection because, according to the post-mortem report, the right recurrent was found eroded and, therefore, the possibility cannot be denied that the fibers supplying the adductors were precluded or paralysed from the beginning.

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* This law does not extend, of course, to those cases where the nerve is damaged by some injury.

(*a*) **Central Paralysis.**—As has been said in the General Section of this Part (see p. 362), the innervation of the larynx extends to the cerebral cortex. Here we may find two centers for the voluntary functions of the larynx, viz., one center for adduction (center of phonation) well developed, and a second center less well developed, for abduction (center of forcible respiration). The centers ruling the involuntary functions, viz., reflex production of voice, as in coughing, emotion, and quiet respiration (automatic center of phonation and respiration), lie in the medulla oblongata; and here, contrary to the case of the cortical centers, the abductor center prevails (center of quiet respiration).

1. *Cortical paralyses* are almost always functional and are usually due to hysteria; organic lesions of the cortical centers are only very exceptionally observed (see below).

(*a*) *Hysterical Paralysis of the Laryngeal Muscles* (*Aphonia Hysterica*).—*Etiology and Pathology.*—*Both cortical centers* of phonation are, as a rule, affected, and the automatic centers in the medulla remain unaffected. Hence the paralysis only concerns *the adductors*, inasmuch as they are subject to *the will.* The patient is *able to speak*, but *without voice*, but *cough*, however, remains loud. Respiration, which is governed by the medulla, is undisturbed.

It very seldom happens that the abductors, viz., all the muscles supplied by the recurrent nerve, are also paralysed. "Hysterical paralysis of the posticus" is, perhaps, nothing but an example of inspiratory functional spasm of the glottis (see p. 587), or a disturbance of coördination of a functional nature.

Women are mostly, but not exclusively, the subjects of hysterical laryngeal paralysis; and they lose their power of speech usually as the result of strong emotion or injury. Hysterical aphonia may be reflexly brought about by disease of neighbouring organs, and also by the agency of the genital apparatus. It is, therefore, not infrequently found during puberty and pregnancy, at the climacterium, in cases of displacement of the uterus, in cases of nasal polypi, hypertrophy of the lingual tonsils, and in pharyngitis lateralis.

Symptoms.—Hysterical paralysis usually sets in suddenly and disappears almost as suddenly as it has started, a sign that it cannot be due to an organic lesion. In some cases it only suffices to rapidly compress the thyroid plates several times (*Olliver's* seizure), or to introduce a mirror or even an instillation in order to at once restore speech. Sometimes, however, this result is not achieved without a few spasmodic attempts on the part of the patient. The patient always complains of various sensations,—so characteristic of hysteria,—and also shows other signs of hysteria (hysterical stigmata). In certain cases, however, there is nothing but aphonia. In severe cases the centers for articulation are affected, and the patients are then not only voiceless, but they are also unable to whisper ("mutismus hystericus," hysterical muteness, apsithyria). The reflex voice-sounds are preserved: that is, the patients are able to cough loudly, cry, laugh, and sometimes even to sing.

Laryngoscopic Examination. (See Figs. 204, 205.)—The glottis forms a triangular space on an attempt at phonation, the base of which is often very small. Not infrequently the vocal cords exhibit a jerky movement towards the median line, but are able to go back rapidly into the "respiratory position." In some cases the ventricular folds are pressed together, while the vocal cords, only visible near the posterior laryngeal wall, show a distinct gap between them. *Hysterical paralysis of the laryngeal adductors is always bilateral.*

Diagnosis.—This must be based on the rapid change from the normal voice to aphonia, on the full sounding cough and other signs of hysteria, and on the laryngoscopic examination, viz., *bilateral paralysis or paresis of the adductors.* In young girls and women who have lost their voices suddenly and without apparent cause the diagnosis "hysterical paralysis of the adductors" can be made at first sight (par premier coup).

Prognosis is favourable, but there are usually frequent relapses.

Treatment must, above all, be directed against the cause. The paralysis usually disappears quickly if the larynx is compressed, as described above, or painted. Intralaryngeal faradisation also works wonders. Whatever method we use, the patient must be exhorted to at once intonate after our manipulation, and must be required to speak a few words, or otherwise the voice will again fail. Practice in speaking may be suitably combined with exercises in respiration. It is frequently advantageous to take the patient by surprise, so to speak, with the treatment, as the unexpected psychic influence is the best remedy.

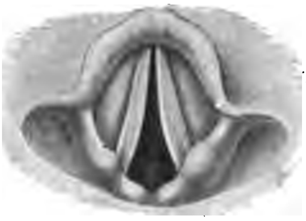

Fig. 204.—Hysterical paralysis of the adductors on phonation.

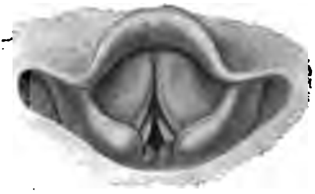

Fig. 205.—Hysterical paralysis of adductors. Vicarious adduction of ventricular folds. Small gap between the posterior ends of vocal cords (phonation) (*Chiari*).

(*b*) *Paralysis of the Larynx from Organic Cortical Disease.*—Bilateral organic lesions of both laryngeal cortical centers are possible, as, for instance, in the so-called pseudo-bulbar paralysis, where the causes of the disease, such as haemorrhage and gummata, are situated in the brain, either in the cortex cerebri or in the corona radiata, *i. e.*, among the fibers running from the cortex to the medulla. Such symmetrical conditions, however, are of the rarest occurrence, and if they should really occur, they take so rapid a course that a paralysis of the larynx probably always escapes discovery.

Paralysis of both cortical centers of respiration has not yet been observed, and, furthermore, it would not be of any practical importance, for quiet respiration remains undisturbed.

Unilateral lesion of a laryngeal center in the cortex is unimportant and does not much matter. This harmonises with the fact, ascertained by experiment, that the exclusion of one cortical laryngeal center on one side only produces no signs whatever, as the center for the other side at once assumes the double function (see p. 362).

2. *Bulbar or bulbo-spinal paralyses of the larynx* may be caused by a whole series of morbid conditions, *e. g.*, haemorrhage, gumma, tumour, progressive bulbar paralysis, amyotrophic lateral sclerosis, syringomyelia, and locomotor ataxia. Here, in the root area of the laryngeal nerves, the above-mentioned *Semon-Rosenbach's* law prevails (see p. 557), which says that in organic lesions the abductors only are affected, and in progressive disease also the abductors are affected prior to the adductors.

It, therefore, consists of a destruction of the bulbar center of abduction, either unilaterally or bilaterally. If the disease advances, the center of adduction may become involved. Commencing occasionally with a paralysis of the "posticus," often after some years have elapsed, a paralysis of all the laryngeal muscles may supervene. In short, an incomplete paralysis of the recurrent nerve becomes ultimately complete. According to the site and extent of the primary cause, the paralysis of the laryngeal muscles may be isolated or combined with that of other cranial nerves.

(*b*) **Peripheral Paralysis of the Larynx.**—Considering how large is the area over which the peripheral neurons of the vagus are distributed, and which brings this nerve into close relation with very important organs or parts of organs, we ought not to be surprised to find how great is the variety of factors which may interfere with the function of the laryngeal nerve. We specially refer to the processes in the vicinity of the nerve which, by their results, interfere with the nerve in a lesser or greater degree. On the other hand, disease of the recurrent nerve itself (neuritis, perineuritis) may lead to interruption of its functions. In the former case we have to deal with tumours chiefly, aneurysms, and inflammatory processes of the larynx, trachea, and neighbouring organs. In the latter mostly with infectious diseases, such as diphtheria, influenza, and poisoning by lead, arsenic, etc. Catarrhal processes and phlegmonous inflammations, and also any overstraining of the voice, are liable to affect the nerve. It will, however, be difficult, in such cases, to decide whether the nerve or the muscles alone, or both together, are affected, the last being certainly the more frequent occurrence. Finally, there may be injuries causing direct lesions of the nerve.

In all these cases of peripheral paralyses of the vagus above mentioned, paralysis of the superior laryngeal nerve excepted, *Semon-Rosenbach's* law holds good, and we see that, just as in organic lesions of the laryngeal centers, the nerve-fibers to the "posticus" are the first to be paralysed (see above). The paralysis may concern the vagus nerve-trunk or the superior laryngeal or the inferior laryngeal nerve, or only branches of them may be affected.

1. *Paralysis of the Trunk of the Vagus.—Etiology.*—The nerve may be damaged immediately after its exit from the brain, and the vagus is here so intimately connected with the spinal accessory nerve that both nerves may be affected by one and the same lesion.

Whether the accessory nerve or the vagus is the motor nerve of the larynx—and on this question we have already touched (p. 353)—we cannot here consider at length. Experiments (chiefly *Grabower's* investigations) have decided this question in favour of the vagus. The clinical observations which are supposed to prove the case for the spinal accessory—we refer to the paralyses of the sterno-cleido-mastoideus and trapezius muscles in combination with laryngeal paralysis—are explained by *Grabower,* who assumes that the pathological process causing the vagus to degenerate spreads from this nerve towards the accessory. This assumption is based on the supposition that all cases of reported paralysis of the accessory nerve are peripheral. In any case a central lesion, viz., degeneration of the accessory nuclei, has not been demonstrated, and those cases, therefore, in which the paralysis of the two muscles supplied by the spinal accessory nerve was combined with a laryngeal paralysis are of no clinical value, because they were not proved by post-mortem examination. On the other hand, three cases supported by post-mortem investigations show that in central lesions accompanied by laryngeal paralyses the roots of the vagus have been found degenerated, while the accessory nuclei remained intact. This goes to prove that the vagus should be considered as the motor nerve of the larynx.

The causes which may lead to paralysis of the vagus are tumours, such as sarcoma or carcinoma, gumma, or inflammations at the base of the skull, and injuries. In the further course of the nerve there may also be tumours, injuries, goiter, inflamed lymphatic glands, aneurysms, etc.

Symptoms depend on the exact spot at which the nerve is affected. In lesions *above* the branching off of the superior laryngeal nerve all the laryngeal muscles of the same side are paralysed, and there is anaesthesia of the corresponding half of the laryngeal mucous membrane, and the pulse-rate is greatly increased, even up to 160. In lesions *between* the origins of the two laryngeal nerves there is no anaesthesia, but only paralysis of the muscles. In lesions *below* the recurrent the larynx remains intact, while the cardiac symptoms may be very alarming. If, in addition, the pharyngeal branches are paralysed, then we shall also find paralysis in the pharynx and anaesthesia of the palate.

Unilateral paralysis of the vagus is also liable to produce symptoms and signs of paralysis or spasms on the other side. Whether this occurs reflexly or by an ascending degeneration of the paralysed vagus spreading to the nuclei of both sides is still an open question.

2. *Paralysis of the Superior Laryngeal Nerve.—Etiology.*—Isolated paralysis of the superior laryngeal nerve is rare, and is

almost always found only as a sequela of diphtheria. It must be regarded as an ascending neuritis, which also usually occurs in other peripheral nerves. There is mostly, in addition, a paralysis combined with anaesthesia of the soft palate (see pp. 304, 333, et seq.).

Symptoms.—The laryngeal mucous membrane is insensitive. The anaesthesia is only absent in the cases where the external branch of the laryngeal nerve is alone affected (see p. 353). On the other hand, it might be possible, in a lesion of the trunk of the superior laryngeal nerve, that the crico-thyroid muscle may escape paralysis if this muscle is supplied by the inferior laryngeal nerve, as sometimes happens (see p. 353). Unilateral paralysis of the crico-thyroid muscle manifests itself laryngoscopically by the corresponding vocal cord being seen to be below the level of the vocal cord of the unaffected side during phonation. Some muscular fibers also which are attached to the epiglottis and act as a depressor of the epiglottis are likewise said to be occasionally paralysed.

The clinical symptoms which must be referred to laryngeal anaesthesia have been discussed previously. (See p. 353.) The voice sounds impure and rough, if the crico-thyroid muscle, which serves as a tensor of the vocal cord, is paralysed. High-pitched sounds become difficult, and speech soon exhausts. If one places the tip of the forefinger into the space between the thyroid and cricoid cartilages, one may distinctly observe that the usual approximation of the cricoid to the thyroid cartilage, on attempts to produce high-pitched sounds, is wanting.

The reports of cases of paralysis of the superior laryngeal nerve certainly require further supplementation, on account of the singularity of this form of paralysis.

3. *Paralysis of the Inferior or Recurrent Laryngeal Nerve.*—*Etiology.*—Paralyses or pareses of the recurrent nerve are frequent, and are due to the same agencies which also affect the trunk of the vagus, but the recurrent, comparatively, is much oftener paralysed than the vagus, owing to its exposed position. The causes have been roughly outlined already (see p. 562); they are:

1. Diseases in the vicinity, *e. g.*, aneurysm of the aortic

arch (on the left side) and of the innominate and subclavian arteries (on the right side); pathological processes at the apex of the right lung; carcinoma of the oesophagus; less frequently, goiter; swelling of lymphatic glands; mediastinal tumours; pleurisy; and pericarditis.*

2. Injuries following operations or caused by a stabbing affray or attempted suicide, etc.

3. Neuritis and perineuritis, in the wake of infectious diseases; and chronic poisoning by lead, arsenic, alcohol, etc.

In some cases the cause cannot be found, and one then resorts—*faute de mieux*—to catarrhal paralysis. That central lesions also may lead to complete or partial paralysis of the recurrent has already been stated.

According to the seat of the lesion, the paralysis is either unilateral or bilateral. It is *complete* if the trunk of the recurrent nerve is damaged; or *incomplete,* if only single fibers or branches thereof, and accordingly single muscles or groups of muscles, are paralysed.

(*a*) *Paralysis of a Single Adductor.*—1. *Paralysis of the M. Thyro-arytenoideus Internus.*—This form, shortly termed "paralysis of the internus," is very frequent; it is the most common of all single muscle paralyses, and is usually met with in catarrhal laryngitis and as the result of overstraining of the voice; it is also common in hysteria. Of the adductors, the "internus" is doubtless the most susceptible. It is the first of the adductors to strike work after the "posticus," if once a progressive organic lesion of the larynx has begun.

The symptoms comprise a more or less severe disturbance of voice, and in marked cases there is aphonia.

Laryngoscopic Image.—The vocal cord appears concave and somewhat more slender. In bilateral paralysis the glottis forms an oval or elliptic chink during phonation. (See p. 433 and Figs. 206, 207.)

2. *Paralysis of the M. Arytenoideus Transversus.*—Paralysis of the "transversus" is much less frequent than that of the

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* Paralysis is hardly ever due to mere pressure on the nerve. The thin nerve usually escapes to a certain extent. It is probably always due to inflammatory processes which have spread from the tumour to the nerve, and destroy its structure either directly or by adhesions and contraction.

"internus," with which it is often combined, but it is yet more frequent than paralysis of the "lateralis" (see below). Disturbance of the voice is usually very marked, but, curiously enough, it is altogether missing in some cases.

Laryngoscopic Image.—The hindermost part of the glottis, viz., the pars intercartilaginea, gapes in the form of a triangular space, the apex of which is directed forwards. (See Figs. 208 and 209.)

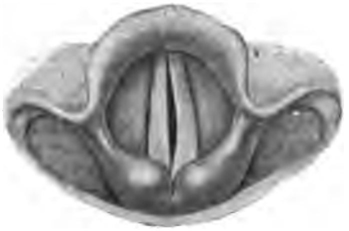

Fig. 206.—Paralysis of left m. thyro-arytenoideus internus during phonation.

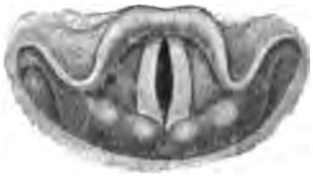

Fig. 207 —Bilateral paralysis of m. thyro-arytenoidei interni during phonation.

3. *Paralysis of the M. Crico-arytenoideus Lateralis.*—Whether or not an isolated paralysis of the "lateralis" ever occurs is still a matter to be questioned. The vocal cord in such a case would remain more or less laterally from the median line during phonation, and the glottis would gape and so cause severe hoarseness or aphonia.

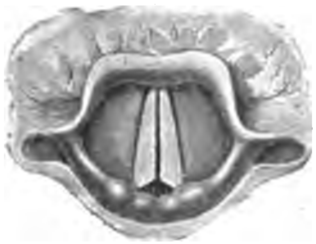

Fig. 208.—Paralysis of the M. arytenoideus transversus during phonation.

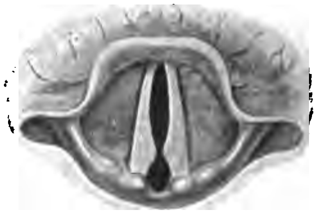

Fig. 209.—Paralysis of the M. arytenoideus transversus and both mm. thyro-arytenoidei interni (combined paralysis of "internus" and "transversus") during phonation.

(*b*) *Paralysis of all the Adductors.*—This is probably always due to central (cortical) lesions of the functional order, as in hysteria, and has been discussed in the chapter on Central Paralyses. (See p. 558.)

(*c*) *Paralysis of the Abductors.*—Let us recall the fact that abduction of the vocal cords is the act of the m. crico-arytenoideus posticus sive "posticus." Accordingly, if only one muscle alone or both muscles are paralysed, we speak of unilateral or bilateral paralysis of the "posticus."

The cause of this form of paralysis must be sought for in slow, progressive organic changes in the medullary centers or in lesions of the trunk of the vagus itself. In the former case tabes dorsalis is the most common cause, and here the paralysis of the "posticus" forms a complication *sui generis.* In the second case, according to *Semon-Rosenbach's* law, it represents the initial stage of a total paralysis of the recurrent nerve, viz., a partial or incomplete paralysis of the recurrent nerve. In tabes, however, many years may elapse before the paralysis of the recurrent nerve will be complete. *A long-standing paralysis of the "posticus" is, therefore, very significant of locomotor ataxia.*

If the "posticus" is paretic or paralysed, the outward movement of the vocal cords is inhibited. The more the paralysis advances, the less the abduction becomes, and the more the vocal cords approach the median line. Finally, the vocal cord becomes fixed in the median line ("median position").

Let us see how this position is brought about. Relying on the *Schmidt-Schech's* experiments, it is assumed that, in the case of the "posticus" being paralysed, the adductors draw the vocal cords towards the median line, and keep them fixed in the adducted position. This would then amount to a contracture of the adductor antagonists. Opposing this opinion, *Kuttner* points out that not only in bulbar (tabetic), but also in peripheral, paralysis of the "posticus," *destructive* lesions are usually combined with *irritative* ones. Spasm of the adductors is added to the paresis of the abductor, on account of the irritation of the corresponding center. These spasmodic contractions of the adductors tend more and more to draw the vocal cords towards the median line and to keep them in this position, the more the pathological changes which affect the "posticus" advance. If such spasms are repeated, or if they persist for a shorter or longer period, then changes occur in the ligamentous and muscular apparatus of the vocal cords, according to *A. Kuttner*, which finally fix the vocal cords in the "median position."

1. *Unilateral Paralysis of the "Posticus."*—The clinical symptoms in one-sided paralysis are insignificant. The patient does not complain of any trouble, and the paralysis is often only discovered by mere chance. Setting aside those rare cases of paralysis of the "posticus" in children, respiration is usually sufficient so as not to cause dyspnoea. The phonation is altogether unaltered, because the adductors and tensors required for phonation remain intact, at first, at least, and later on the action of the m. crico-thyroideus alone is quite sufficient to render tense the vocal cords, which are fixed in the "median position," so that intonation can take place correctly.

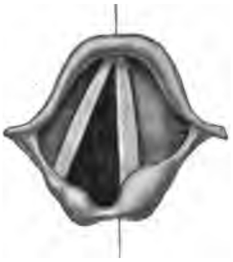
Fig. 210.—Paralysis of left crico-arytenoideus posticus during respiration (*Réthi*).

Laryngoscopic Image.—During respiration the glottis forms a triangle, one side of which is pretty well in the middle line, and during phonation the usual narrow chink is formed. (See Fig. 210.) *This picture may be observed to remain for months and years without alteration.* As the paralysis advances, the adductors begin to fail—first, the "internus" (p. 565); later on, perhaps, the "transversus" also; so that one may at last see the picture depicted in Fig. 209. If, finally, all the fibers of the recurrent nerve become paralysed, the vocal cord assumes the "*cadaveric position,*" which, however, does not correspond to the actual position in corpses, but to an intermediate position between phonation and respiration.* Thus the paralysis of the "posticus" may become changed into a complete paralysis of the recurrent nerve.

2. *Bilateral Paralysis of the "Posticus."*—In opposition to unilateral paralysis, double paralysis of the "posticus" is a very serious affection, which may even endanger life by urgently

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* The width of the glottis post-mortem is not constant. Rigor mortis has a great influence upon the position of the vocal cords.

threatening suffocation. Since a double paralysis occurs only in tabes, the resulting stenosis developes very slowly, so that the patients become gradually accustomed to a reduced air-supply, in such a way that, at first, they only become dyspnoeic during bodily efforts. In due course, however, the inspiratory dyspnoea increases (see p. 385) and may assume such a degree that only a very slight incident, such as a catarrh, will prove sufficient to occasion the most serious danger. (See p. 408.) It is remarkable, however, that expiration remains unchanged in spite of the stenosis. The voice sounds remain normal, or almost so, because the vocal cords are entirely or nearly in the "median position" requisite for phonation, but sometimes the voice is somewhat impaired.

The symptoms may be complicated by the paralysis developing in an asymmetrical manner, *e. g.*, as in carcinoma of the oesophagus.

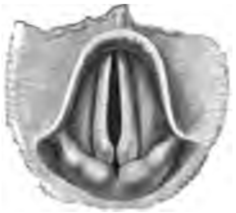

Fig. 211.—Paralysis of both the crico-arytenoidei postici. Respiration.

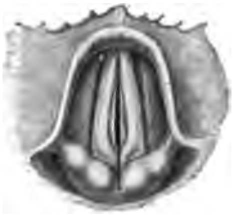

Fig. 212.—Paralysis of both the crico-arytenoidei postici. Phonation.

Laryngoscopic Image.—In marked cases the glottis is narrowed to a fine chink. The vocal cords *during phonation and respiration* remain in the position of phonation, *i. e.*, in the median line, and are approximated so as to touch each other—probably because they are liable to be aspirated towards one another in consequence of the rarefaction of the air in the trachea. (See Figs. 211, 212.) If, later on, the vocal cords assume the "cadaveric position," then the dyspnoea will again diminish, while the voice becomes somewhat more impaired.

(*d*) *Paralysis of all the Muscles Supplied by the Inferior Laryngeal Nerve* (*Complete Paralysis of the Recurrent Nerve*).—We have already, on various occasions, pointed out that complete paralysis of the recurrent nerve is very often only the finale to a paralysis of the "posticus." In slow progressive organic lesions the "posticus" at first refuses to work; then follow the adductors, first, the "internus," and subsequently the other laryngeal muscles, with the exception of the crico-thyroideus, which is supplied by the superior laryngeal nerve, and only occasionally comes into the area supplied by the recurrent nerve. Paralysis of the "posticus" and of the "posticus" and "internus," and of all the muscles supplied by the recurrent nerve, are, therefore, only the various stages of one and the same disease. Under certain circumstances the entire recurrent nerve may be affected from the beginning, *e. g.*, from an injury or wounds.

The so-called "cadaveric position," which the vocal cords assume in total paralysis of the recurrent nerve, may be considered, so to speak, as the position of "indifference," created by the failure of both groups of muscles, *i. e.*, narrowers and dilators of the glottis.

One must here also discriminate between unilateral and bilateral paralysis.

1. *Unilateral Total Paralysis of the Recurrent Nerve.*—This is more frequently observed than the bilateral form. The most important, if not the only, symptom is the alteration of the voice. Since, in long-standing paralysis, the healthy vocal cord compensates for the loss of action caused by the paralysed vocal cord (see below), the voice does not usually sound very hoarse, but only somewhat rough, impure, or "cracked," and may assume some kind of double sound (diphonia) during forced speech, owing to the unequal vibrations of both vocal cords; but, on the other hand, it may run into a falsetto. The voice sounds somewhat weak (phonasthenia) and soon becomes tired. Both these symptoms may be explained by the wasting of the air-blast, on account of the inhibited closure of the glottis. (See p. 385.)

Massei has recently shown that paralysis of the recurrent nerve is accompanied by defects in laryngeal sensibility. Should his observations prove true, then it ought to demonstrate that the recurrent nerve was not purely motor, but a "mixed nerve." Experiments on animals do not permit the same conclusions with regard to man. In dogs, the recurrent nerve is doubtless a motor nerve only, possessing a borrowed sensibility for a short distance. In other animals, such as in apes, cats, rabbits, and goats, it also contains sensory fibers.

Laryngoscopic Image.—The paralysed vocal cord is in the "cadaveric position" during both respiration and phonation. The arytenoid cartilage, owing to paralysis of the "posticus," whose action it is to draw the cartilage backwards and outwards (see p. 568), stands a little forwards and is tilted slightly inwards. (See Figs. 213, 214.) It also appears to be larger, and the vocal cord seems shortened (because it appears to be hidden behind the cartilage).

In long-standing paralysis the vocal cord atrophies; it becomes slender, smaller, and somewhat concave. In recent paralysis the healthy vocal cord during phonation approaches the median line, and forms, with the other (paralysed) vocal cord, a triangular space situated somewhat towards the paralysed side. During respiration it goes back into the abducted position, and the glottis gapes towards the healthy side. In paralysis of long standing the healthy vocal cord during phonation trespasses on the median line, thus approaching the paralysed vocal cord, and effecting a more or less complete closure of the glottis. In this way the glottis assumes a somewhat *oblique* position, and the *arytenoid cartilages appear to be crossed.* (See Fig. 215.) The arytenoid cartilage of the paralysed side stands in front of the

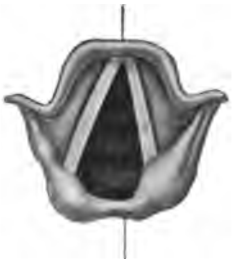

Fig. 213.—Paralysis of left recurrent nerve (during respiration). Left vocal cord in "cadaveric position"; right vocal cord abducted (*Réthi*).

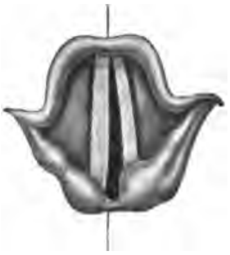

Fig. 214.—Paralysis of left recurrent nerve (during phonation). Left vocal cord in "cadaveric position"; right vocal cord adducted to "median position" (*Réthi*).

cartilage of the healthy side, and therefore the image appears to be above the healthy cartilage. (See p. 406 and Fig. 215.)

In some cases of recent paralyses the arytenoid cartilage, immediately after phonation, shows jerking, oscillating movements towards the median line. These jerky movements are probably caused reflexly by contraction of the single musculus "transversus," which still obtains its impulses from the healthy side. Jerky movements of the epiglottis have also been observed.

If the paralysis abates, the adductors first recover, and, later on, the "posticus," whose susceptibility is so great that it is not only affected earlier, but also remains affected for a longer time.

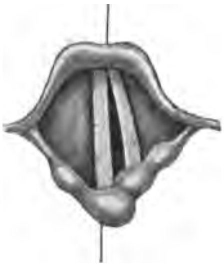

Fig. 215.—Paralysis of left recurrent nerve (during phonation). Left vocal cord in "cadaveric position"; right vocal cord has traversed the median line; the arytenoid cartilages show "crossing"; the paralysed cartilage stands in front of the healthy cartilage (*Réthi*).

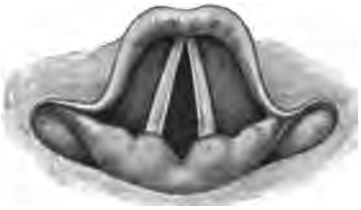

Fig. 216.—Bilateral paralysis of the recurrent nerve symmetrically on both sides.

Thus the total paralysis again becomes a paralysis of the "posticus."

2. *Bilateral Total Paralysis of the Recurrent Nerve.*—This is far less frequent than the unilateral total paralysis, because the primary disease—injuries excepted—has most probably resulted in death before both recurrent nerves have become paralysed.

Laryngoscopic Image.—The paralysis is not always equal on both sides. One may find total paralysis on one side, viz., "cadaveric position" of the vocal cord, and on the other side paralysis of the "posticus" alone, viz., "median position"; or the motor lesion may be very pronounced on the one side, and only faintly expressed on the other side. In total paralysis of both sides the glottis gapes during phonation and respiration in the "cadaveric position." (See Fig. 216.)

The vocal cords, having once become atrophic, tremble during respiration, and are passively moved and aspirated towards each other, so that a closure of the glottis may be effected. Aphonia is the chief symptom. The patient cannot cough well nor can he clear the throat. The permanently open glottis permits a great waste of the air-blast, and even renders whispering a great effort, which soon fatigues the patient. There is no dyspnoea, as the air-passages remain open, except in very asymmetrical paralysis. Particles of food may easily find their way into the larynx or lower air-passages, and thus may lead to death from pneumonia.

Diagnosis.—In making a diagnosis, we must answer three questions: (1) Is there a paralysis or paresis? (2) Which muscles or group of muscles are paralysed? (3) What is the cause of the paralysis?

1. *Is there a paralysis or not?* The most important criterion of laryngeal paralysis lies in the inhibition of mobility; and this can only be discovered by the mirror.

Occasionally, anamnesis and clinical symptoms may be of value, but disturbances of voice and respiration per se don't usually mean much. One might perhaps assume a bilateral paralysis of the "posticus," if there were inspiratory stridor combined with dyspnoea, while the expiration and phonation remained free or nearly so. Then one might suspect a double paralysis of the crico-thyroidei, if the patient is not able to produce high-pitched sounds, and if the space between the cricoid and thyroid cartilage does not then diminish. (See pp. 564 and 565.) This sign is naturally of little value in persons with short, fat necks.

If an inhibition of mobility has been discovered by the mirror, we have to prove further that it is due to paralysis, and was not *eventually caused by some mechanical hindrance.* The processes in the superficial and deeper layers of the mucous membrane, already discussed on various occasions, here play an important rôle. Swellings of the mucous membrane and infiltration on the posterior laryngeal wall, or developing posteriorly between the vocal cords; ankyloses and pseudo ankyloses of the arytenoidal joints; scars, etc., are prone to inhibit the movements of the vocal cords. In such cases it would probably not be very difficult to note the hindrances. In other cases, however, they are so insignificant that they do not offer much help in forming a diagnosis.

It is well to remember that in paralyses, at least in the neuropathic paralyses, no pathological changes which would serve to explain the palsy show themselves on the laryngeal mucous membrane.

2. *Which muscles or group of muscles are paralysed?* In order to estimate and interpret correctly the motor lesions seen by the mirror, it is necessary to thoroughly know the functions of the muscles and their associated actions.

3. *What is the cause of the palsy?* Here, at once, great difficulties may arise, and a general examination of the entire body may be necessary. Very often only the presence of many symptoms together will indicate to us the right track. On the other hand, the laryngeal palsy may be just the only symptom, perhaps initial symptom, of an aortic aneurysm,* a carcinoma of the oesophagus, a mediastinal tumour, or tabes. Unfortunately, with all our cunning, we may not always be able to make a diagnosis, and only the course of the disease eventually teaches us that we should not be too ready with our diagnosis of "rheumatic palsy" in a case where we could not find any other cause.

(As for hysterical palsy, see p. 558.)

Prognosis.—*Quoad vitam,* the double paralysis of the "posticus" and the palsy of the *superior* laryngeal nerve are serious. The former may at any moment give rise to severe dyspnoea; the latter constantly threatens the danger of pneumonia from misswallowing. *Quoad restitutionem ad integrum,* the primary cause determines the ultimate course and ending. If this be irremediable, then there can be no chance of curing the affection. For this reason cortical or bulbar processes are unfavourable. Only the syphilitic and hysterical palsies prove an exception, though the latter are often quite incalculable. Tabetic palsies also sometimes show a glimpse of improvement, inasmuch as we may succeed, according to *Goldscheider's method,* in training the muscles or nerve-fibers which remain intact.

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* In aortic aneurysm the typical form of palsy is the paralysis of the recurrent nerve, and this may be explained by the topographic relations of the aorta to the recurrent nerve.

Of the peripheral palsies, those caused by aneurysms, oesophageal carcinoma, or mediastinal tumours have a bad prognosis. Post-diphtheritic paralysis, however, often subsides if a pneumonia from misswallowing (see above) does not previously finish off the case. Catarrhal palsies usually subside along with the laryngitis, though they often show themselves more obstinate than the catarrh.

In the hypokineses from unknown causes, one should be careful in one's prognosis, for it not infrequently happens that a "rheumatic paralysis" later on turns out to be not so harmless as at first seemed to be the case.

Treatment should always, if possible, be directed against the primary cause of the paralysis, but, unfortunately, in most of the central paralyses, and in many of the peripheral paralyses, we are not able to influence the primary causative disease. (For the treatment of hysterical paralysis see p. 560.)

In syphilis, mercury ointment and iodide of potassium: of the former, 3 to 4 grains of blue ointment; of the latter (10 : 200), a tablespoonful three times a day should be given. Less successful, but well worth trying, is iodide of potassium in aneurysm or goiter pressing on the nerve, remembering that aneurysm is sometimes caused by syphilis and goiter often diminishes under the influence of iodine. In those cases also of unknown cause iodides might be tried, because there may be some swollen glands which may benefit from this treatment. Under stress of circumstances gelatin injections in the cases of aneurysm would be justifiable. Post-diphtheritic paralysis requires general roborant treatment and feeding by the oesophageal tube in cases of marked laryngeal anaesthesia. Catarrhal processes must be treated as previously described (pp. 450 et seq.).

The paralysis itself must be treated by the electric current, mostly by the faradic, and less frequently by the galvanic, current. At the beginning of the treatment both electrodes can be applied outside on both sides of the larynx; later on the electrodes may be introduced inside the larynx, after previous local anaesthesia, in order to increase the effect. (See p. 394.) The patient, however, very soon becomes used to the intralaryngeal electrification, so that one may then dispense with the local anaesthesia. According to *Ziemssen*, each muscle has its own point of stimulation. (See Fig. 217.)

Fig. 217 shows these motor points. I myself do not think that it is possible to stimulate each muscle separately, as intralaryngeal faradisation always has a general effect, *i. e.*, it causes closure of the glottis, and soon some retching also. Under such circumstances it would be very difficult to place the electrodes exactly on the particular spots indicated. If one wishes to electrify the inferior laryngeal nerve, it is sufficient to place the electrode into the lower segment of the pyriform sinus, *i. e.*, corresponding to the beginning of the oesophagus. It would hardly be possible to reach the nerve from the outside. On the other hand, one easily succeeds in stimulating the crico-thyroid muscles, as their "irritative" points are situated at the front of the neck, in the space between the cricoid and thyroid cartilages, on both sides of the median line.

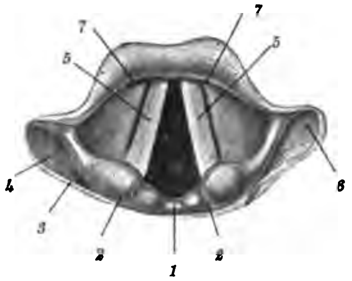

Fig. 217.—Points of stimulation of the laryngeal muscles (after *Ziemssen*).

The current can be applied externally, every second day, for three to five minutes at a time; but if intralaryngeally, then for a shorter space of time. During electrisation and afterwards, the voice should be gently exercised by requesting the patient to intonate vowels and to repeat syllables uttered by the physician. On the whole, we must not expect too much, especially in long-standing paralysis. Intralaryngeal electrisation should be omitted altogether in cases of aneurysm, so as not to cause accidental rupture of the aneurysmal sac from retching. In double paralysis of the "posticus" tracheotomy must sometimes be performed on account of threatening suffocation, and under certain circumstances this could be done as a prophylactic treatment against suffocation.

II. SPASMS (HYPERKINESES).

The spasms, tonic rather than clonic, are caused by irritative processes which excite the motor nerves of the larynx either directly, from central or peripheral causes, or indirectly and reflexly. In the latter case they are spoken of as reflex neuroses.

(A) **Laryngospasm (Spasm of the Glottis).**—Laryngospasm is a spasmodic seizure of the glottis, which must be regarded as part of a general respiratory cramp. The laryngeal muscles are involved in the spasms of the other muscles of inspiration and expiration. Laryngospasm, however, is due to spasmodic contraction of the adductors alone, which, from their number and mass, predominate over the abductors, so that the latter muscles are overpowered.

As the centers for adduction and abduction lie close to each other in the cortex, as well as in the medulla, and, owing to the fact that the laryngeal muscles—with the exception of the crico-thyroid muscle—are all innervated by the recurrent nerve, one would expect, a priori, that every stimulus acting on the laryngeal nervous apparatus, centrally or peripherally, would produce spasm of all the laryngeal muscles. This is, indeed, the case with only few exceptions (see below). In children, in particular, where the different centers are not yet quite distinct, all the laryngeal muscles will always be excited together. Laryngospasm in children is much more frequent than in adults, and also shows clinically some differences. We shall, therefore, discuss both forms separately.

(*a*) **Spasm of the Glottis in Children** (*Laryngismus Stridulus*).—*Etiology.*—The disease occurs in children of from six months old to two years of age, who are badly nourished or overfed. This circumstance harmonises with the fact that nine out of ten children affected by laryngospasm are subject to rachitis. It is not the rickets per se or the softness of the occiput (craniotabes is, however, only a symptom of rickets) which causes the spasm, but the faulty nourishment of the artificially fed children, which is admittedly the cause of rachitis. In the last significance it is always a faulty decomposition in the intestinal tract, viz., an autointoxication, which excites the spasms; but, of course, this occurs only in children predisposed to it, that is, in overexcitable, hypersensitive children. With regard to this, laryngospasm stands in the same rank as tetany and convulsions, for it is frequently only a forewarning of one or other of these conditions of general spasm. Very slight incidents are sufficient to excite spasm of the glottis in such overexcitable children, *e. g.*, "swallowing the wrong way" while drinking, depression of the tongue for the purposes of examination, etc. Obstinate children, if they do not at once get what they want, almost wantonly throw back their heads, and at once fall into convulsions, though often only slight attacks. The parents admirably describe this as "going off" into a fit.

Among the central causes of laryngismus stridulus, subacute and chronic hydrocephalus plays a not unimportant rôle.

Symptoms.—In mild cases the respiration pauses only for a moment, and is resumed with a long-drawn-out hiccuping or crowing inspiration. The peculiar high-pitched sound is produced by the spastically adducted vocal cords being set in vibration by the forced attempt at inspiration. During the fit the child becomes pale, but recovers quickly and then cries afterwards, and though the attacks may recur frequently, yet no serious disturbance of the general condition remains. Sometimes the spasm is so slight that it escapes observation.

In the severe cases the symptoms are much more alarming. The respiration stops for a longer time. The child attempts, perhaps, at the beginning of the attack, to draw in air with a hiccuping sound, but the spasm not being relaxed, extends to all the other muscles, and the child, at first flushed, becomes pale and finally cyanotic, rolls its eyes, fixes its limbs (in tonic spasm), clenches its fists with the thumbs turned inwards and with the wrists flexed (carpopedal contraction); it thrusts its head backwards, the abdomen becomes tense, and unconsciousness occurs. The tonic respiratory spasm may then pass on into clonic spasms of the limbs of shorter or longer duration. This is called "infantile eclampsia," whereby consciousness is suspended; froth may often be seen in the mouth.

In the most severe cases such an attack may result in death, and the child then succumbs to paralysis of the heart. But more frequently the respiration, having stopped for a few seconds or even for a minute, the child again recovers. The limbs and body begin to jerk, and a few crowing sounds, repeated at short intervals, announce the entrance of air into the larynx. The spasm of the larynx relaxes, and respiration becomes again quiet. The child still remains exhausted for some time after the fit, but finally recovers from it completely. The fits occur, as a rule, without any warning, and are repeated at shorter or longer intervals; some are mild, others more serious. The disease may last for months, viz., as long as the overexcitability and disorder of the intestines are present.

Diagnosis.—In pronounced cases the diagnosis will not be difficult. The absence of cough and fever, as well as the sudden onset and short duration of the attack, will prevent confusion with diphtheria or other organic disease of the larynx. Distinction from pseudo-croup (see p. 435) or whooping-cough is sometimes possible only after longer observation. In double paralysis of the "postici," *constant dyspnoea* is apparent. Foreign bodies may also excite attacks of laryngospasm.

Congenital Stridor Laryngis.—Laryngismus stridulus must be distinguished from *congenital stridor laryngis:* the infants, often from the first day of life, make stridulous noises, as if from dyspnoea. It is assumed that in such a case it consists of some kind of disturbed coördination—of a short clonic spasm of the glottis, which ends with an expiration. But recent investigations have shown that an enlarged thymus or an abnormal development of the aditus laryngis must account for the mechanically produced stridor. It is certain that this kind of stridor is not due to true stenosis, and, accordingly, cyanosis is never found, as it is in true spasm of the glottis. The unpleasant symptom after a time almost always subsides spontaneously.

Prognosis.—Laryngismus stridulus rarely leads to death, though it does sometimes happen in severe cases. Frequent repetition of the fits, in conjunction with the failure of nourishment, keep the children back and may weaken them so much that they may easily fall a prey to intercurrent diseases.

Treatment.—It does not often happen, and only if the attacks are of frequent occurrence, that the physician will witness such an attack, but if so, the child should be erected, the windows thrown open, and respiration may then be excited by splashing the chest of the child with cold water, or, still better, by artificial respiration. A tepid bath with cold affusion also acts well. If the spasm persists for a long time, the forefinger may be inserted into the mouth as far as the epiglottis, and the epiglottis lifted by depressing and pulling forward the tongue.

According to *Kürt*, stimulation of the *trigeminal nerve* reflexly causes inspiration. It is said that one can cut short the attack by touching the conjunctiva or tickling the nasal mucous membrane. This would tend to prove that the trigeminal nerve is an inhibitory nerve. Treatment might perhaps benefit by this theory, if it were to be shown that cauterising the nasal mucous membrane with silver nitrate or trichloracetic acid were able to prevent the attacks.

In very severe cases all efforts are in vain: neither intubation nor tracheotomy will save the child, for though these operations certainly afford air enough, still they can exercise no influence upon the paralysed heart.

Treatment should aim at the causes, viz., at the elimination of the disorders of nourishment, *e. g.*, rachitis; and at mitigating the overexcitability. Careful feeding, therefore,—if possible, by wet-nurse,—fresh air, saline bathing, and purgatives (calomel, as an intestinal disinfectant, 0.01 gm.; sacch. lactis, 0.3 gm., thrice daily); and when the dyspepsia has subsided, then cod-liver oil with phosphorus (ol. jecoris aselli, 100.00 c.c.; phosphor., 0.01 gm., one teaspoonful twice daily), and a nerve tonic, should be given for some weeks. In mild cases the tinctura valeriana aetherea can be given in 5- to 10-drop doses in sugar and water three or four times daily; or bromides:

℞. Sod. bromid.,	
Ammon. bromid.	āā 1.0–1.5
Aq. dest.	ad 100.00

Sig.—One dessertspoonful thrice daily.

If one desires to act very energetically during a critical attack, one may add to the bromide some chloral hydrate, as follows:

℞. Chlorali hydrati	1.5
Sodii bromid	2.00
Aq. dest	100.00

Sig.—One dessertspoonful thrice daily.

If a fit is expected in the night, and should the administration of medicine by the mouth meet with difficulties, an enema of chloral hydrate could be ordered:

℞. Chlorali hydrati..........................1.00
Mucilag. acaciae..........................50.00
F. M. Sig.—Half to be used for the enema.

(β) **Laryngospasm in Adults.**—*Etiology.*—Setting apart the attacks of laryngeal spasm during or after intralaryngeal manipulations, laryngospasm rarely occurs in adults, but is comparatively most frequent in hysteria. Of peripheral exciting causes, tumours have in some cases been observed, which, under certain conditions, *e. g.*, on change of position of the body, press upon the vagus, but without paralysing it. Other causes are found in laryngeal polypi, which are caught between the glottis, or by foreign bodies flung from the lower air-passages against the vocal cords. Occasionally, pharyngeal and nasal diseases are liable to cause laryngeal spasm reflexly.

Symptoms.—The patient shows all the signs of an acute stenosis. The attack, however, is much less disturbing than in the case of children, apart from the fact that it only occurs very occasionally. Usually some long-drawn-out crowing inspirations can be heard, while the patient anxiously struggles for air. Death results only in very exceptional cases, and under very unfavourable conditions, *e. g.*, if a polypus or foreign body is caught between the vocal cords.

In hysterical laryngeal spasm, other forms of cramps, such as pharyngospasm, tonic or clonic spasms in the limbs, alternate with the laryngeal spasm. This alternation is typical of hysterical spasms.

Diagnosis.—Confusion with bilateral paralysis of the "postici" is not probable, for the vocal cords are permanently closed in paralysis, while in laryngospasm the vocal cords move freely when not in a condition of spasm. To recognise the exciting cause is more difficult; hence one should never forget to examine the entire respiratory tract.

Treatment.—As regards the treatment of spasm occurring during or after intralaryngeal manipulations, see p. 397. For the rest, the treatment must be directed against the exciting cause (tumour, foreign body, nasal or pharyngeal disease, etc.).

(γ) **Laryngeal Crisis in Locomotor Ataxia.**—Laryngeal crises in tabes dorsalis are due to tonic spasm of the adductors of the vocal cords. It here consists of a stimulation of the bulbar center of the adductors, which is a typical symptom of the early stages of the disease.*

The crisis usually begins with a sensation of tickling or tension in the throat; the patient is forced to cough, and this becomes more and more violent; respiration is difficult, and soon the picture of laryngospasm developes, with its long-drawn-out crowing inspirations. The attack may be very mild, but in other cases it is more severe, and is accompanied by giddiness and even unconsciousness; it takes a longer time—about one or more minutes—before the spasm subsides.

Diagnosis.—In laryngospastic attacks in adults it is always well to bear in mind tabetic disease. The differential diagnosis must be made from *whooping-cough*, which may also occasionally occur in adults, and from *ictus laryngis* (see below). *Argyll-Robertson's* symptom (reaction of pupil to accommodation and not to light) and the absence of the patellar reflex (knee-jerks), and *Romberg's* sign are typical of tabes. In the larynx, paralysis of one "posticus," or even of both, is not very rare. This paralysis, which occurs comparatively frequently, and especially in tabes, need occasion no trouble, and is usually due to a progressive degeneration of the medullary centers for abduction. (See p. 561.) We here see in tabes the curious fact that, on the one hand, the center of adduction is stimulated, and, on the other, the center for abduction is paralysed, viz., spasm of the adductors and paralysis (paresis) of the abductors. If, in the course of a paralysis of the "posticus," spasmodic coughing or spasm of the glottis occurs, we may be sure that it is a case of tabes, though the knee-jerks may perhaps be present.

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*Occasionally also the abductors may be affected by spasm, especially in lyssa (hydrophobia). *Pitt* reports a patient suffering from lyssa, who got an attack of spasm during the laryngoscopic examination and showed a maximum dilatation of the glottis for some seconds.

Prognosis in regard to the attacks is good, for they are hardly ever very threatening, and, later on, spontaneously subside.

Treatment.—We can do nothing but diminish the reflex excitability by bromides or inhalations of menthol, and in some cases cocaine proves useful. During the crisis inhalation of chloroform might do good. Frequently repeated attacks or severe crises sometimes require tracheotomy.

(B) **Nervous Laryngeal Coughing.**—*Etiology and Pathology.*—Nervous laryngeal coughing is a species of expiratory spasm. It is characterised by short spasmodic contractions of the adductors and muscles of expiration, and is based on an excessive overexcitability of the coughing center, which is not produced by pathological changes of the respiratory tract, but is almost exclusively the outcome of a general nervous disposition. Psychic and emotional causes, such as fright, rage, shame, embarrassment, etc., play an important rôle as exciting agents; hysteria and neurasthenia, but tabes, epilepsy, and chorea also, are very often the indirect cause. Peripheral stimuli may reach the center by various paths and from various organs.

Thus, after catarrhal laryngitis or tracheitis, the mucous membrane frequently remains very sensitive, which sensitiveness is apt to send stimuli to the center in the medulla by the regular normal way of the superior laryngeal nerve. But stimulations are also carried by other abnormal or unusual paths to the medullary coughing center. Cerumen in the ear or foreign bodies in the auditory meatus, and even the gentlest introduction of an ear-speculum, may provoke paroxysms of coughing. Pathological changes also in the nose (see p. 125), throat, spleen, liver, or genital organs may act in the same manner. On the other hand, pathological changes need not be present in the organs before mentioned; an increased reflex excitability is of itself quite sufficient to elicit coughing on the slightest stimulation. In this way may be explained the coughing caused by masturbation. I remember a case where the slightest touching of the perinaeum was likely to evoke paroxysms of coughing.

The coughing frequently occurs without any visible cause, and in such a case we must assume that the center may have been directly stimulated. Juvenile persons at the time of puberty are those most liable to nervous coughing, for the nervous system at this time of life is particularly irritable and unstable.

Symptoms.—Nervous coughing is preceded by a sensation of tickling, and it rarely occurs in single violent paroxysms; much more frequently it is of a continuous character, so that the patients constantly cough or "clear their throat." The "musical pitch" of the cough described by various authors I could not myself observe, in spite of, or perhaps on account of, my musical sense. The coughing sounds rather like bellowing, bleating, or howling; but all these descriptions are also suitable for the coughing due to laryngitis with scanty secretion. More conspicuous than the timber of the coughing is the circumstance that the patients themselves are little incommoded by their cough, and they certainly suffer less from it than their neighbours, who are sometimes driven to exasperation by the "eternal barking." The patients themselves cannot suppress the cough, and it increases if they feel themselves under observation. The cough stops usually, but not always, during the night, and there is none or only very little expectoration.

By the laryngoscope nothing can be discovered. Hyperaemia, which is sometimes found, is rather the consequence than the cause of the cough. It may, however, be observed that the vocal cords do not close during the cough, a fact which serves to explain why the patients are themselves so little troubled by their own cough.

Diagnosis.—Diagnosis, as the case may be, is not always simple, especially in persons of nervous constitution, with little or no expectoration. In such a case one should not be in a hurry to make a diagnosis, unless one has thoroughly examined the air-passages from the nose downwards. One should remember that whooping-cough, pleurisy, tuberculosis, or even a simple catarrh is prone to keep up the cough for a long time, and that foreign bodies likewise excite coughing. The diagnosis of nervous cough is supported if one finds imperfect closure of the glottis during coughing.

In any case the continuous nervous cough is easier to recognise than that occurring in paroxysms. When the diagnosis is assured, it still remains to ascertain under what conditions the irritation takes place. So the organs mentioned above must be examined as to whether or not there are any pathological changes, viz., nose, throat, ear, womb, etc., and if there are any points of stimulation, *i. e.*, the irritative zones must be carefully made out. (*Cf.* p. 125.)

Prognosis is usually good, although the cough may prove very obstinate, and of a particularly relapsing disposition. If the nervous cough is a part or a symptom of a central disease, such as tabes or epilepsy, the prognosis is naturally bad with respect to these diseases.

Treatment should pay attention to the etiology. If it consists of a reflex neurosis, the eventual changes in the organs concerned must be treated. For the rest, general treatment is the most important. In this respect reasonable hardening, change of air and surroundings, and, according to *Semon*, a sea voyage, and last, but not least, psychical or suggestive treatment, will prove of use. Certain drugs, as iron, arsenic, and bromides, may be given, but the usual "cough remedies" are quite useless.

(C) **Vertigo and Ictus Laryngis.**—There exists a kind of nervous coughing, ushered in by tickling in the throat, which, however, soon leads to actual giddiness or even unconsciousness, so that the patient falls down. The attack usually lasts for a few seconds, and occurs only occasionally, but may do so several times during the day.

Pathology.—The *pathology* of this affection is still obscure. One party regards it as an epilepsy with a special aura in the larynx; another party regards it as a laryngospasm with subsequent syncope. I myself agree with *M. Schmidt*, who assumes that ictus laryngis is the consequence of an acute cerebral hyperaemia due to cerebral congestion.

The course of ictus laryngis has much in common with tabetic laryngeal crises (see p. 582); therefore the knee-jerks and pupillary reflex must be examined.

Prognosis is mostly good, apart from some cases which turn out badly.

Treatment is the same as in nervous coughing.

III. DISORDERS OF COÖRDINATION (PARAKINESES).

Disturbances of coördination are due to faulty innervation of the different laryngeal muscles required for the movements of the vocal cords, in such a way that the voluntary and involuntary (automatic) movements of the vocal cords are performed in an unharmonious manner.

(A) **Phonetic Spasm of the Glottis (Dysphonia sive Aphonia Spastica; Spastic Aphony).**—*Etiology and Pathology.*—Spastic aphonia or dysphonia is a kind of spasm of the glottis which occurs only at the moment of phonation, and it is, therefore, called phonetic spasm of the glottis, in contradistinction to laryngismus stridulus, which latter is not confined to any particular movement, but may also occur during rest. The cortical center of phonation appears to be in a state of irritation, and excites, if stimulated, the muscles to spastic contraction, which more or less exceed the desired physiological effort. According to *Semon*, this state of irritation is caused by overstraining and the faulty use made by professional orators of their vocal apparatus, who speak for a long time continuously without even taking time for breath. Phonetic spasm of the glottis is, therefore, analogous to the mogigraphia (writer's cramp), and must be regarded as a coördinatory occupation neurosis. Besides professional overstrain, hysteria and neurasthenia, and emotion also, play a part in the etiology of the disease.

Symptoms.—On the attempt to phonate, the adductors of the glottis spasmodically contract, and the vocal cords are pressed firmly against each other; so much so that not even the blast of air necessary for the production of voice can pass through (aphonia). In mild cases the patient can utter a hoarse or suppressed sound, but no more than this, as the word actually sticks in the throat. The patient then complains that his neck feels as if strangulated. The disorder gradually developes and increases more and more. At first the speech is like that of a stutterer; the vowels are reduplicated, the diphthongs are divided in their elements, *e. g.*, i–i, a–a, o–u, "I–I–ca-an not spe-e-eak." While, however, the ordinary stutterer is able to overcome the hindrance and speak fluently quite a long series of words, in the case of a sufferer from phonetic spasm of the glottis the disorder becomes aggravated at each attempt at further speech, and it not infrequently happens that other groups of muscles also become involved in the spasm. If the patient ceases speaking, the spasm ends. *Respiration remains free.* Occasionally the spasm may last a little longer, even if the patient has already ceased speaking, so that the following inspiration is somewhat hampered, so that in such a case phonetic spasm of the glottis is combined with "spasm of inspiration." (See below.)

In certain cases the attempt to speak, even in a whisper, to laugh, cough, or to blow out a candle, may excite the spasm.

Laryngoscopic examination shows nothing abnormal beyond the perfect closure of the vocal cords. Not the smallest space can be detected between them, and they appear to be firmly pressed against one another. Not infrequently the ventricular folds are also adducted, and may more or less cover the vocal cords.

Diagnosis is easy from the clinical and laryngoscopic picture.

Prognosis.—The disease is exceedingly obstinate and, as a rule, is not amenable to treatment. I recollect the case of a woman who had been attended for many years, and when I saw her again after thirteen years, suffered from it after that time just as much as ever.

Treatment.—In spite of the slender chance of cure, one should not abandon treatment, but should try to mitigate the disease by hydropathic cures, nervine tonics, and systematic exercises of the voice. *Gutzmann* advises, at first, exercises of inspiration and expiration, and these to be followed by gentle but noisy expirations; then, after these, some exercises in whispering, combined with pressure on both sides of the thyroid cartilage, and, finally, moderate exercises with the full voice.

In some cases the spasm is said to have ceased if a laryngeal probe—after previous anaesthesia—has been inserted between the vocal cords, so that they are prevented from closing.

(B) **Inspiratory Spasm of the Glottis.**—This disorder consists of a "perverse action" of the vocal cords, which approximate at *inspiration*, and thus cause dyspnoea. Expiration and phonation are perfect. Here, also, the origin of the disease must be sought in a kind of overexcitability due to hysteria.

Sometimes the "inspiratory spasm" is combined with an expiratory spasm (see above); the vocal cords approximate during inspiration and are pressed firmly against each other on phonation. In other cases the vocal cords separate on an attempt at phonation, which thus becomes impossible. Hysteria in its protean nature here furnishes a multitude of combinations.

Nervous patients sometimes show at the first laryngoscopic examination a perverse action of the vocal cords: the vocal cords approximate on inspiration and separate on phonation.

Diagnosis.—"Inspiratory spasm" of the glottis might be mistaken for bilateral paralysis of the "postici" (see p. 568), which is also characterised by inspiratory dyspnoea, while expiration and phonation are free. Paralytic dyspnoea, however, is continuous and uniform, whereas the spasmodic inspiratory dyspnoea differs in intensity at different times and disappears during the night. While tabes is usually the cause of paralysis, hysteria is the basis of the spasm. *Semon* advises, in doubtful cases, that the patient should phonate during laryngoscopic examination as long as possible, until finally, from lack of breath, the spasm will be overcome, if only for a moment, and the vocal cords will separate, at least for a few seconds. If there is paralysis, the vocal cords will only close the more firmly, viz., dyspnoea would increase.

Prognosis, as a rule, is favourable in simple inspiratory spasm.

Treatment must be directed against the underlying disease, viz., against the hysteria.

(C) **Other Lesions of Coördination.**—1. *Ataxia of the Vocal Cords.*—It has already been mentioned, in the General Section (see p. 373), that the vocal cords during forced or excited respiration make short rhythmic movements, closing or opening the glottis according to expiration or inspiration. Real ataxic movements, *i. e.*, jerky, irregular movements, occur in voluntary actions, that is, during phonation or forced respiration. Quiet respiration, which is an automatic action, is not subject to ataxia. Such ataxic movements may also occur in tabes, hysteria, bulbar paralysis, and disseminated sclerosis. The jerky movements of the vocal cords seen in chorea are mere concomitant movements.

2. *Nystagmus of the vocal cords*, consisting of rhythmic, oscillatory, or twitching movements, has been observed in hysteria, paralysis agitans, and in neuritis due to chronic poisoning, *e. g.*, lead and arsenic, etc. It is independent of the will.

3. *Prolonged Mutation (Breaking) of the Voice.*—It sometimes happens in boys that the mutation of their voice is not finished within the usual time (see p. 360), but lasts much longer. The voice does not become deeper, but remains in the high register or passes, after acquiring a few low tones, into a falsetto (pathological castrate or falsetto voice). The cause is a disturbance of coördination due to a faulty use of the phonetic muscular apparatus.

Treatment is very often powerless in face of marked predisposition to the affection. Pure hysterical disturbances recover under proper treatment, but are prone to relapse.

Prolonged mutation requires methodic exercises in speaking; the patient must be required to speak with a deep voice a few syllables or words; later on, these are followed by sentences.

XI. AFFECTIONS OF THE VOICE IN SINGERS AND ORATORS.

Etiology.—There are a great multitude of causes which may, in due course, lead to impairment of the voice, and hardly any affection of the air-passages leaves the voice entirely unaltered or undisturbed. Singers and orators are subject to the same conditions as other mortals who do not require their voice exclusively for trade or professional purposes. We will, therefore, only discuss here those factors which are likely to interfere with the professional use of the voice, and, at the same time, consider the distinction between the *singing* and the *speaking* voice.

The singers' art requires a specially coördinated action of the various muscular groups. A lesion in any part of the vocal apparatus will, therefore, be noticed much sooner comparatively by the audience or by the singer himself than is the case with the orator, who is certainly able to minimise slight changes much easier than can the singer. There is, further, a difference between a correctly trained voice and one faultily or inadequately trained. A singer or orator who knows how to produce and use his voice will be better able to stand a strain and do so for a longer time than a less experienced artist or an adept who has not yet completed his studies and is, therefore, not yet sufficiently trained.

Finally, but not the least important, it always is the straining of the voice beyond the limits of its natural power that alters the voice temporarily or permanently.

Overstrain may be due to intralaryngeal or extralaryngeal causes. To the intralaryngeal causes, above all, belongs catarrh of the laryngeal mucous membrane, as is so often found in smokers or drinkers and persons who are compelled to sing or speak under bad hygienic conditions. The extralaryngeal causes are: diseases in the region of the resonator (mouth, pharynx, and nose) and of the lungs, which serve the purpose, so to speak, of the bellows, and disorders also of the digestive tract; general weakness from anaemia or convalescence from illnesses, etc. Those disorders of the voice which occur in connection with certain conditions of evolution or decay of the body are still, partly, at least, within physiological limits. Here we may mention puberty, which, however, does not much concern singers and orators, as the training of the voice only usually begins after the voice has fully developed. More important, however, is menstruation, although this can only temporarily influence the voice. The laryngeal mucous membrane and also that of the resonating tube may become more loose and congested during the time of menstruation, with the result that the voice becomes sooner tired and reduced in volume at the upper end of the register.* These disturbances are naturally greater in cases of dysmenorrhoea. Pregnancy exercises the same influence as menstruation. Finally, advancing age, by reason of ossification of the cartilages and increasing atrophy of the muscles, alters the sound, vigour, and elasticity of the voice. This process of involution sometimes manifests itself in women, sometimes even prior to the climacterium; but more frequently it is at the "change of life" that the paraesthesias of the larynx and pharynx, as also other nervous disorders of these organs, are apt to alter the voice. After that time the voice may again recover its former freshness and colour, and retain them for a long time.

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* As a rule, songstresses should be excused from performing, and pupils of musical colleges should not attend practice of singing, during the time of menstruation.

Faulty training is often responsible for permanent damage to the voice. Too long exercises, or those that do not correspond to the register of the voice, faulty technique, leading to abnormal position of the larynx, which inhibits natural breathing, and the correct formation of the resonance chambers are all apt to overstrain the voice and so to seriously derange it.

Lastly we must mention the disorders of the voice which are neuropathic in character. Nervous singers or orators are often "indisposed," and show differences in their vocal capacities. It is just the stage artist who, knowing that his fame and gain are dependent on his voice solely, so easily becomes a throat hypochondriac, and who is so excited at the slightest irregularity, often, indeed, only fancied, that he loses control of his voice. In other cases, again, it is "stage fright" which acts in an inhibitory manner.

In hysteria the derangement of the voice assumes the character of a hyperkinesis, hypokinesis, or parakinesis. (See p. 556, et seq.)

In many cases various causes are busily at work.

Symptoms.—At first only certain subjective symptoms indicate that there is anything wrong with the larynx, for there may be a sensation of "tiredness" or paraesthesia and pain in that region. In some cases the sufferers concerned may discover an existing alteration in their voices before the trained ear of any one else could detect anything wrong, or before the laryngoscope was able to reveal any change. If, however, the alteration of the voice has existed for a prolonged period, then the results of the affection of the voice may also be objectively observed in the sense that the defect is noticeable not only to other persons, but to the singer or orator himself.

The *accuracy* and *purity* of the voice are the first to suffer. The singer misses the right note, not because he hears falsely, but because he faultily tenses the vocal cords. He "discords," sings out of tune, especially when changing his register or on intoning high notes, or when singing softly (piano). Impurity of the voice should be distinguished from "discording." In "discording" the tone is higher or lower than the desired tone, by half a tone or by a still smaller interval which is not determinable musically; and the note achieved is false in relation to an accompanying instrument, but is in itself pure; whereas if the voice is impure, the tone itself sounds impure, on account of the associated noises produced by the simultaneous vibration of catarrhal secretions. In such a case the voice sounds impure—"veiled."

The accuracy of tone also very often suffers along with the *sustentation* of the voice. The note cannot be held without wrongly merging into a higher note, or still oftener into a lower note, and loses also in intensity (power). This decrease of intensity may be gradual, until the voice entirely fails, or the tone sounds alternately weaker and fuller. The last instance is spoken of as "trembling," or "tremolo," and usually also slight rhythmic irregularities here play an important part in regard to the height of the note. Tremolo is very often only a technical fault, due to bad habits or bad training, or to mechanical causes, such as catarrhal secretions or swellings of the vocal cords. In some cases, however, it represents only a special form of mogiphonia, where, as has been said previously (see p. 556), the voice suddenly fails with a painful sensation of "tiredness," the larynx revealing nothing pathological on examination.

The *timbre* of the voice very frequently suffers. This kind of alteration is produced by faulty configuration of the resonator, *i. e.*, nose, mouth, and pharynx; either by faulty muscular action from bad training or habit, or from pathological changes which alter the shape or the spaciousness of the cavities. Thus the sound of the voice assumes another character: *e. g.*, it *becomes thick*, muffled, and colourless if the lining membranes of the nasal (or pharyngeal) cavity are thickened, or if the space is diminished by polypi; or it *becomes oral* (palatinal) if the velum palati is contracted and too much elevated; or choked and "throaty," if the muscles between the jaw and hyoid bone are too much contracted, thereby dragging the larynx too far up. This alteration of the voice is spoken of as being "guttural in tone," in contradistinction to the "dental" tone which occurs if the mouth is not opened enough and consequently the vibrations are checked by the teeth. In this sense, also, badly fitting artificial teeth may have the same effect. In any case, whether the alteration of the voice is produced by a faulty technique or by anatomical conditions, it always leads to overstrain and subsequently to becoming soon fatigued and exhausted while singing or speaking.

The changes observable laryngoscopically are dependent on the duration of the derangement. We mainly find a picture of chronic laryngitis in its various stages. At first we only find the secretion increased, and here and there small lumps or threads of mucus may be seen. Later on hyperaemia occurs, and, finally the vocal cords—the one sometimes more than the other—appear reddened and swollen. The "singer's nodes" previously described (see p. 449) only represent a special form of laryngitis. In old-standing cases the vessels of the vocal cords become dilated, and finally pareses occur, which latter may, however, appear before the signs of chronic laryngitis are fully developed.

The pharynx shows similar changes, and lateral pharyngitis is especially frequent.

In many cases *nothing can be seen by the mirror*, though one's ear can distinctly detect the alterations of the voice. Whether such an alteration is due to a mere muscular insufficiency or is also associated, exclusively perhaps, with a disturbance of innervation, is not easy to decide. In general, as has been pointed out (see p. 556), one must assume that the disorders of mobility are not due to an insufficiency of muscular effort alone, but that there are always also lesions in the nervous mechanism.

Diagnosis.—Above all, one must acertain whether there is an actual derangement of the voice, and, if so, what is the cause of it.

The question whether there is an actual derangement may be answered by the mirror, if, indeed, one discovers anatomical changes in the larynx (catarrh, "singer's nodes," pareses, etc.). Frequently, however, the laryngoscopic examination fails to reveal any abnormality in the larynx. Changes are absent or are not so fully developed that they can be noticed, for actors and singers, as a rule, consult the physician promptly and at a very early stage of their voice derangement. Hence one must not allow oneself to be guided entirely by the laryngoscopic examination, *but should test by ear the voice of the patient.* For this, of course, a trained musical ear is necessary.

It would be of diagnostic value to test the conditions of the vibratory capacity of the vocal cords during the examination. The laryngeal mirror is not suited for this purpose, because its insertion and the pulling out of the tongue alter the shape of the resonating tube so much that the timber of the voice becomes quite changed. *Oertel* has advised for this purpose his laryngostroboscope, which permits the observation of the vocal cords during vibration.

The cause of the vocal disturbance is not always easy to discover, and by no means always at the first examination. Suppose one has found the vocal cords reddened, as one frequently sees in singers or orators within certain limits, and the anamnesis, which is in many cases of immense value, fails to explain in our particular case the cause of the redness; then one must certainly not decide from the one examination solely as to whether we have to deal with hyperaemia from mere overactivity as a sign of muscular strain, or whether there is actual laryngitis. The decision, however, can be made after two or three days, having, in the mean while, ordered the patient to rest his voice during that period. Hyperaemia from overaction will have entirely subsided by this time, while a catarrh, especially if chronic, will show little or no change. If one has decided that there is a catarrh, one has further to show whether it is entirely confined to the larynx, or whether it is continued from any other organ (pharynx, naso-pharynx).

The examination of the resonating tube is, therefore, very important. If one discovers nothing there, then one must examine the general state of health, and, above all, the nervous system. If the nervous system, on thorough examination, proves to be intact, then we may infer that the disturbance of the voice is due to a bad habit or to faulty training.

Prognosis.—This is the more favourable the earlier the disturbance of the voice is recognised and the earlier it has been attended to. Unfortunately, people belonging to the artistic world are distinguished by their carelessness, and frequently their inevitable material and artistic worries or engagements, interfere very considerably with their chances of recovery.

Generally speaking, the prognosis will depend on what the patient expects of his art and his art requires of him. A chorister and a music-hall singer need have less fear than perhaps a "vocalist," "prima donna," or "hero-tenor," and an actor values his voice higher than a teacher, preacher, or member of parliament or congress.

The changes of the voice produced by involution, and which in some cases appear very early, afford, of course, a bad prognosis. Here all treatment is powerless, and everything depends on how much and how long the orator or singer is able to compensate or cover up the defect in his voice.

Treatment.—Attention must be chiefly directed to the causes of the derangement. In this respect hygienic and general treatment are very important. We must here, therefore, apply the same rules as have been already considered necessary for the proper care of the *healthy* voice. (See p. 402.)

If the disturbance of the voice is produced by a faulty technique or faulty method of instruction, then the teacher must be changed as soon as possible. Unfortunately, the most important demand—that each voice should be developed individually, according to its individual conditions, often remains unrealised, owing to the carelessness or insufficient knowledge of the teacher, and frequently, also, through stress of the material worldly position.

If the derangement of voice is due to overstrain, the larynx must be rested for a time, and rest is also demanded in cases where one finds signs of redness or inflammation. It might be difficult to say whether the redness and swelling of the mucous membrane are the cause or the consequence of overstrain, but wherever they are found, the vocal apparatus urgently requires rest. It would perhaps be best to forbid singers and actors to appear before the public as long as there are signs of inflammation. However, in order not to bring the artist into too great a conflict with his contracts by our draconic orders, one may occasionally make a concession by allowing an "indisposed" singer to perform his duty if we find the vocal cords white, though there may be signs of irritation.* One may give such permission to experienced singers or actors who are able to control their vocal apparatus and temper according to the advice of the physician. One may also, however, do much in order *to help the artist safely through the performance.* Menthol lozenges are very useful, as has already been mentioned on various occasions, and one may be taken shortly before the commencement and two or three during the rest of the performance. If the patient complains of dryness in the throat, Soden or Ems pastilles are very serviceable, or the well-known sal ammoniac pastilles or pastilles d'Orateur, which enjoy general favour. Should the nose prove to be obstructed, it may be advisable to paint the nasal mucous membrane shortly before the performance with a 1 per cent. adrenalin solution, and afterwards with 5 per cent. cocaine solution. These drugs may also be handed to the artist so that he could, if need be, repeat the painting once or twice during the evening.

In acute catarrh of the upper air-passages all "professional use" of the voice must be strenuously forbidden, and the catarrh itself must be treated in the ordinary way, and the patient told that the prescription must be strictly followed, owing to the great delicacy of the vocal apparatus. Total rest may be succeeded later by a period during which the singer may be allowed to gently practise once more, and the actor also must only gradually resume his recitations. This transient period lasts from one to three days, according to the severity of the disturbance, and during it the singer is only allowed to sing or to recite sotto voce, and with long pauses.

Local treatment in acute catarrh should be avoided under any circumstances, and in chronic catarrh one should make it a principle to interfere only if there are indications for local manipulations.

Operative measures in the larynx of singers or actors are not justifiable unless urgently needed, and painting with concentrated

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* In exceptional cases, we find, in a healthy larynx, vocal cords which show a slightly reddish colour.

solutions is altogether inadmissible. The vocal apparatus of the artist, which is used by him in so subtle a manner, and is capable of the highest artistic performances, is liable to suffer, only too easily, a permanent loss of its capability by operative interference. I use as an astringent in such cases 2 to 3 per cent. silver nitrate, and this only in cases of inveterate chronic catarrh. Inhalations are of no value, for they act more upon the pharynx than on the larynx; they are even apt to relax too much the laryngeal mucous membrane, but in certain dry forms of laryngeal catarrh they exercise a very beneficial action.

Anomalies of the resonating tube should, as has been previously pointed out, be carefully looked for. But here also it must be our guiding principle not to do too much, not to be "too active," and, more especially, in the nose, which is so frequently, *sit venia verbo*, the happy hunting-ground for operative busybodies, not to sacrifice each little corner, or each little projection. A nasal cavity that resonates well is indispensable for a singer or orator, but the dilatation of the nasal lumen must not be too exaggerated, in order not to prepare the way for ultimate atrophic changes, which are apt to alter the timbre of the voice considerably. The removal of large tonsils, dreaded so much by singers and actors, is advantageous, with regard to the health of the voice; but a lesion of the palatine arches would involve a great mistake, for the scars that afterwards remain may, according to *Flatau*, permanently influence the production of voice by their retraction.

If insufficiency of the laryngeal muscles has become manifest, and if pareses have developed, the electric current may be applied extralaryngeally or intralaryngeally, provided that there is no other organic disease. The therapeutic value of the electric (faradic) current is, however, *not* to be exaggerated, as also in other (non-laryngeal) motor lesions, and the same may be said of massage. Systematic exercises appear to be of greater value, and they may be practised if all pathological changes have subsided. These exercises consist of methodically practising the singing and speaking voice, which, however, must be very rigidly adapted to individual propensities. At the same time, faulty technique in the production of voice and speech, which would serve as a cause of derangement of the voice, must be corrected.

According to *Bottermund* and *Spiess*, exercises should be commenced with practice in whispering, thereby fostering the coördinate articulation and vocal muscles. Exercises in whispering are followed by voice exercises, with alteration of the words; at first, mezzoforte, and in moderate intervals of thirds and fourths in a suitable register of voice—men with chest or middle voice, women at first only with middle voice, and always avoiding the limits of their registers. In practising the middle voice, and later on the head voice, the vowels should be properly nasalised, *i. e.*, the resonance of the head made good use of.

In cases of great weakness of the voice *Bottermund* inserts between the whispering and vocal exercises the practice of humming, *i. e.*, he advises the patient to sound "m" with the mouth shut, and in a conveniently high tone.

Insufficiency of the laryngeal muscles in orators, according to *Bottermund*, is still more quickly cured by the practices above mentioned than that of singers. The reason for this is because the essential feature of systematic training of the voice, viz., the learning of the necessary breathing economy, with regard to the desired blast of air for the vibration of the vocal cords, affords greater rest to the laryngeal muscles during speech. The objection is made that only musical persons are suited to such methods of practice, but the objection, however, is untenable, as it does not matter in principle whether the vocal exercises should be artistic or not.

However this may be, the exercises must be repeated daily and for a long period, but the patient must never continue to sing until he or she is tired.

APPENDIX.

MENINGITIS CEREBROSPINALIS EPIDEMICA.

Etiology and Pathology.—The naso-pharyngeal space plays an important rôle in the etiology of epidemic cerebrospinal meningitis. The carrier of the disease, either the diplococcus pneumoniae (*Fränkel*) or *Weichselbaum-Jäger's* diplococcus intracellularis meningitidis (meningococcus), is said to penetrate into the cavity of the skull from the pharyngeal tonsil, viz., the adenoid tissue situated behind the nose. *Westenhoeffer* maintains that the hypertrophy of the pharyngeal tonsil, as generally also a lymphatic constitution, predisposes to the acquisition of cerebrospinal meningitis. The pharyngeal tonsil has thus the same importance in regard to the meningococcus as has the palatine tonsil in regard to the streptococcus. *Weichselbaum*, *Ghon*, and *Jäger* have had the opportunity of showing that the meningococcus was also in the nasal secretions of healthy individuals who had been in contact with persons suffering from meningitis. Such persons, without being themselves ill, are, therefore, able to transmit the disease (*e. g.*, by coughing, sneezing, or spitting) to persons who are predisposed to it. It is similarly the case with diphtheria.

The disease of the naso-pharyngeal space will be difficult to prove for the simple fact that the meningitis usually runs a very acute course, and because it very often affects children, who cannot be or are only examined with difficulty. *Westenhoeffer*, in the postmortem examination of his cases, has nearly always found an acute and severe inflammation of the pharyngeal tonsil and adjacent mucous membrane, while the anterior portion of the nasal cavity was usually found to be free in children, but affected in adults. These statements are corroborated by *E. Meyer*. During the great epidemic in Silesia (1905) acute pharyngitis was ascertained, intra vitam, in the majority of cases.

According to *Westenhoeffer*, the germs advance by way of the lymphatic vessels into the maxillary cavity, sphenoidal sinus, cavum tympani, and cranium; but are never found in the ethmoidal cells. The frequency of diseases of the accessory cavities in epidemic cerebrospinal meningitis may thus be understood. The disease becomes manifest if the cocci have established themselves within the skull and have there caused purulent meningitis.

Whether the meningitis is or is not caused by disease of the ethmoidal cells, perhaps oftener than *Westenhoeffer* assumes, possibly through the lamina cribrosa, may be left for further controversy. Besides the lymph-vessels, the blood-vessels also play a rôle in the conveyance of the germs, as may be seen by the general infection occurring so often in the course of the disease.

Prognosis is dependent on the "genius epidemicus."

Treatment.—Setting aside the few reports of success by the treatment of the disease with meningococcus serum, treatment does not show much success. Lumbar puncture, praised so highly when it first became public, fails or has only a temporary effect, while internal remedies are as good as useless. *Westenhoeffer* prefers to treat the disease according to surgical methods, like other suppurations, and suggests the opening of the skull and drainage of the meninges. He thinks this justified on the ground that if hydrocephalus is once established, it leads either to death or idiocy.

Prophylaxis has a difficult task to perform. To render innocuous the healthy propagator of the pathogenic germs is a pium desideratum, and is easier said than done. It has been pointed out that mines, with their greater humidity, higher temperature, and want of sunlight, are a favourable breeding-place for the saprophytic meningococci, and special measures have been devised in this respect. Effective prevention in practice could be carried through in such a way that, by systematic and repeated examination of the naso-pharyngeal space and of the secretions, the carriers of infectious germs could be discovered and properly taught that they must avoid coughing or spitting or sneezing in the face of others; that they should disinfect their nasal and postnasal cavities by gargles and sprays; and disinfect likewise the sputum, etc.

The value of these disinfecting measures is, of course, not very great, and, therefore, the disinfection of the sick-room should be confined to the neighbourhood of the sick-bed, and this the more so as the meningococcus appears to soon perish from the splendour of its surroundings, even without special disinfection.

INDEX.

PAGE.

- Abscess of larynx 442
  - peritonsillar 266
  - retropharyngeal 268
  - of septum nasi 62
  - of tongue 184
  - of tonsil 266
- Acarus folliculorum (see *Comedo*) 50
- Accessory cavities of the nose, anatomy of 6
  - cysts 162
  - foreign bodies in 164
  - inflammation of 132
  - injuries of 164
  - new growths 162
  - parasites in 164
  - syphilis of 165
  - tuberculosis 165
- Acheilia 181
- Acne rosacea, of nose 53
  - vulgaris 50
- Actinomycosis of mouth 214
  - of nose 114
- Actino-therapy 553
- Adam's apple 342
- Adenoid, habitus 287
  - tissue 236
  - in nasopharynx 236
  - vegetations 283
- Adenotomy 289
- Adhesions (see *Synechiae*) 55
- Adrenalin 22
- Ageusia 220
- Air, current of, in respiration 13
  - deflation of 23
  - inflation of 24
- Alcohol 46, 405
- Alveolar sinus 6
- Alypin 22
- Amyloid tumor of larynx 530
- Anaemia, artificial 21
  - laryngeal, in tuberculosis 480
- Anaesthesia, artificial (local), of larynx 553
  - artificial, of nose 124
  - general 553
  - paralytic, of larynx 399
  - paralytic, of mouth 222

PAGE.

- Anaesthesia, paralytic, of nose 21, 41
  - paralytic, of pharynx 331
- Anaesthesin 22
- Aneurysma aortae 409, 412, 414, 574
  - paralysis of vocal cords in 409, 414
  - tracheal stenosis in 409, 412
- Angina, aphthous 260
  - catarrhal 260
  - dental 260
  - follicular 262
  - habitual 260
  - herpetic 272
  - lacunar 262
  - Ludwig's 227, 268
  - phlegmonous 267
  - Plaut-Vincent's 474
  - post-operative 80, 260, 440
  - pre-epiglottic 183
  - pultaceous 300
  - scarlatinous 297
  - syphilitic 312
  - tonsillar 262
- Angioma of larynx 530
  - of lips 216
  - of nose 119
  - of pharynx 321
- Angioneurotic oedema of larynx 440
- Ankyloglossum 182
- Ankylosis of arytenoid joint 466
- Annulus migrans 193
- Anomaly. See *Malformations.*
- Anosmia 121
- Anthrax 212
- Antrum of Highmore 147
- Aphonia in hysteria 558
  - laryngitis 387
  - spastic 586
- Aphthae, Bednar's 199
  - epidemic 188
  - stomatitis from 187
- Aprosexia 286
- Apsithyria 559
- Articulation, disturbance of 175, 177
- Arytenoid cartilage, anatomy of 343
  - overcrossing 406

PAGE.
Arytenoid cartilage, perichondritis........................ 460
Asthma, bronchial............. 128
  differential diagnosis of 91
  in hay-fever.......... 90
  treatment of.......... 91
Ataxia, locomotor............. 588
  of vocal cords......... 588
Atresia of choanae............ 56
  of nose............... 56
Autolaryngoscopy............. 498
Autophonia................... 336
Autoscopy.................... 378
Autumnal catarrh............. 91

Bednar's aphthae............. 199
Beta-eucaine.................. 22
Bier's treatment.........73, 226, 251
Blandin-Nuhn's gland 172
Bleeding from adenotomy...... 292
  from larynx.......... 389
  from nose. See *Nose bleeding.*
  from polypus of septum 119
  from tonsillotomy..... 295
  from tracheotomy..417, 420
Bougie, treatment of larynx by dilators of tin............... 429
Breaking of voice (see *Mutation*). 361
Bronchial stenosis..........411, 514
Bronchoscopy, direct.......379, 516
  inferior......... 382
  superior........ 380
Brushing of larynx............ 395
  nose............. 40
  pharynx.......... 253
Bulbar paralysis.............. 331
  paralysis of soft palate in anaesthesia.......... 553
  paralysis of larynx...... 561
Bulla, ethmoidal...........5, 7, 153
Burning of larynx............. 524
  mouth 183
Bursa, pharyngeal..........236, 275

Cacosmia...............32, 123, 134
Cadaveric position of vocal cord 568, 570, 572
Calcification of cartilages (ossification)..................... 590
Canula, chimney-shaped, in tracheotomy..................... 430
Carcinoma of antrum.......... 163
  of larynx.......... 540
  of lip............... 216
  of nose............. 119
  of pharynx......... 327

PAGE.
Carcinoma of polypoides........ 544
  of tongue........... 218
  of trachea.......... 552
  ventricular.......... 544
Caries, dental............36, 183, 286
Castrate, voicé of.......387, 405, 589
Cataplasma, treatment by, in laryngeal and pharyngeal disease....................... 251
Catarrh, ascending............. 431
  descending..........36, 431
  upper air-passages..... 431
Catheterism of larynx and bronchi......................... 426
Cauterisation of larynx......... 397
  nose........... 40
  pharynx....... 253
Centre of phonation.........363, 558
  respiration........363, 558
Chamaeprosopia............... 83
Chancre. See *Primary sclerosis.*
Chest voice.................. 361
Chicken-pox in throat......... 467
Children, fixation of, in examination..... 112
  in operation 112
Choanae, atresia of............ 56
  hook for operation.... 118
  polypi............... 325
Chorditis, inferior. See *Laryngitis subglottic.*
  superior. See *Prolapse of ventricle.*
Chorea, lesions of coördination in 589
Cleaning the nose.............. 38
Cleft lip...................... 180
  palate.................... 255
  uvula 255
Climatic treatment............ 249
Clothing...................... 45
Cocaine....................22, 400
Comedones.................. 50
Concha...................... 81
Condyloma in larynx........... 503
  mouth........... 209
  nose............ 100
  pharynx......... 313
Congelatio 51
Contusion of larynx........... 522
Coördination, lesions of, in chord. 588
  in larynx.. 586
Copper nose. See *Nose.*
Corpora cavernosa, congestion of 12, 102, 125
Coryza, acute................ 69
  chronic............... 74
  neonatorum 100
  nervous.............. 127

PAGE.

- Coryza, professional 70
  - vasomotorial 127
- Cough in hysteria 559
  - in laryngitis 359, 387
  - nasal 129
  - nervous 388, 584
  - in tumor of the larynx ... 531
  - whooping 468
- Crista septi 57
- Croup 470
- Curettage in laryngeal tuberculosis 496
- Cynanche gangrenosa 228
- Cysts, laryngeal 529
  - lingual 217
  - maxillary 162
  - pharyngeal 321
  - upper maxillary 162

- Decanulement after tracheotomy 420
  - difficult 420
  - final 421
- Decubitus from canula 421
- Detuning 593
- Deviation of septum 57
  - treatment of 59
- Diaphragm of larynx 406
- Digital examination 29
- Dilatation of larynx 429, 510
- Diphtheria, complications of 304
  - laryngeal 469
  - nasal 93
  - oral 204
  - paralysis in .305, 333, 563
  - pharyngeal 300
  - sensory lesions ..331, 554
- Diphthonia 387, 531
- Diplophonia 387
- Dislocation of septum nasi 58
- Disorders of swallowing 109, 246, 252, 332, 334, 384, 512, 545, 553
- Diverticle of pharynx 257
- Douche, nasal 38
  - precautions 38
- Duct, Bartolini's 173
  - naso-lachrymal 5
  - Rivinus 173
  - Stenonian 172
- Dysmenorrhoea, disturbances of voice in 590
  - nasal disturbances 129
- Dyspepsia in nasal diseases 35
- Dysphagia in carcinoma 543
  - in perichondritis 384
  - in tuberculosis 481
- Dysphonia, spastic 586

PAGE.

- Ear affections in adenoid vegetations 283
  - nasal disease 333
  - paralysis of soft palate 333
  - pharyngeal synechia 257
  - rhinitis, acute ..35, 71
  - rhino-pharyngitis acuta 259, 263
  - rhino-pharyngitis chronica 275
- Eclampsia infantum 578
- Eczema of introitus nasi 47
  - of vestibule 47
- Electric treatment in diseases of larynx .. 394
  - paralysis of larynx .. 575
  - paralysis of pharynx 254
  - paralysis of soft palate 335
- Empyema of accessory cavity 134, 147
- Enchondroma, laryngeal 530
- Enuresis nocturna in adenoid vegetations 286
- Epiglottis 341
  - erection of 361
  - omega-shaped 370
  - retroflexed 370
- Epulis 217
- Epistaxis. See *Nose bleeding.*
- Erb's plaques scars 191, 209
- Ethmoidal cells, anatomy 8
  - inflammation . 140
- Eucaine 22
- Eustachian tube 233, 279
- Exophthalmus 120, 148
- Expectorants 392
- Eye in complications of nasal disease 35

- Facies leonina in leprosy 108
- Falsetto 361
- Felt sponges 253
- Fibro-adenoma of nose 115
- Fibroma, laryngeal 527
  - nasopharyngeal 323
  - throat 322
  - tongue 217
- Foetor ex naso from foreign bodies 33
  - leprosy 108
  - malignant tumor 163
  - malleus 107
  - ozoena 85
  - scleroma 105

PAGE.
Foeter ex naso syphilis......... 100
Foetor ex ore 33, 178, 248, 328
  concrements of tonsils............ 279
  diphtheria....... 204
  mercurial........ 200
  scurvy........... 199
  stomaca......... 197
  stomatitis, aphthous.......... 188
  ulcero-membranosa........... 220
Foot and mouth disease........ 188
Foreign bodies in accessory cavity......... 164
  bronchi....... 513
  larynx........ 513
  nose......... 109
  pharynx...... 318
  trachea....... 513
Fracture of larynx............ 525
  nasal bone........ 61
  septum............ 61
Frenulum of tongue, shortness. 181
  too long... 181
Frontal sinus. See *Sinus.*
Furred tongue................ 178
Furunculosis, diabetic.......... 490
  vestibuli......... 49

Galvanocautery in larynx ... 401
  nose...... 42
  ulceration . 536
Gargles...................... 252
Gargling..................... 252
Genital organs and nose........ 128
Gingiva. See *Gums.*
Gingivitis..................... 182
Glanders in larynx............. 510
  mouth............. 212
  nose.............. 107
  pharynx........... 315
Glandula, Blandin-Nuhn's...... 172
  parotitis 172
  sublingual........... 172
  submaxillary........ 172
Glandular fever, Pfeiffer's...... 226
Globus hystericus..........218, 336
Glossitis, chronic superficialis... 190
  (see also *Macroglossia*). 194
  Moeller's............ 194
  parenchymatous...... 194
  syphilitic............ 209
Glossodynia................... 222
Glottis...................... 347
Glottis, spasms of, in adults.... 336
  in children .. 577
  omega shaped 406
Goitre, diving................ 415

PAGE.
Gonorrhoea of nose............ 93
Gottstein's tamponade........37, 88
Graminol.................... 91
Gymnema acid............... 221

Hair tongue, black............ 192
Hajek's hook................. 160
Hardening.................... 45
Harelip 180
Hay-asthma.................. 90
Hay-fever................... 90
Head voice................... 361
Heart neurosis and nose........ 129
Hemiglossitis, herpetic......... 186
Herpes....................... 272
Hiatus, semilunar.............. 6
Hoarseness in laryngitis........ 388
Hydrargyrosis................ 209
Hydrocephalus................ 578
Hydrops of antrum............ 162
  of frontal sinus ...147, 162
Hydrorrhoea, nasal..........34, 127
Hypaesthesia. See *Anaesthesia.*
Hyperaesthesia, larynx......... 554
  nasal.......... 124
  oral........... 222
  pharynx....... 332
Hypergeusia.................. 221
Hyperkeratosis of pharynx..... 317
Hyperosmia.................. 123
Hypertrophy, lateral. See *Laryngitis Chronica.*
Hypogeusia.................. 220
Hypopharyngoscopy........... 245
Hypopharynx................. 244
Hyposmia 121
Hysteria, paralysis in.......... 335
  sensory lesions in..124, 555
  smell................ 122
  spasms in 223, 336, 581
  taste 221

Ictus laryngis................ 585
Infantile nodes of vocal lips..403, 453
Infiltration, glanders...107, 212, 315, 510
  leprosy 108, 212, 316, 510
  scleroma ...105, 314, 508
  syphilis 100, 207, 312, 503
  tuberculosis 96, 205, 309, 482
Influenza, accessory cavities.... 70
  nose............... 93
  paralysis of.......... 333
  soft palate in........ 469
Infundibulum................ 6
Inhalation................252, 398

PAGE
Injury of accessory cavity...... 164
  larynx.............. 522
  nose................. 61
  pharynx............ 259
Insufflation into larynx........ 397
  nose.......... 40
  pharynx....... 253
Intracranial complication in diseases of accessory cavities..... 36
Intubation................422, 510
  complications of..... 425
Iodide of potash............... 104
Iodipin....................... 105
Iodism....................... 104
Iodovasogen.................. 427

JOINT affections. See *Larynx*.

KIERSTEIN's method of examination. See *Autoscopy*......... 17
Kieselbach's locus.........53, 65, 85
Killian's method of examination. 20
Kiotomy..................... 283
Koplik's spots in measles ...203, 297

LACTIC acid.................99, 192
Lamina cribrosa and sphenoidal sinus...................... 159
Lamina cribrosa, perpendicular. 3
Larvae of flies................ 113
Laryngeal crises...........553, 582
  examination in. See *Laryngoscopy*.
Laryngismus stridulus.......... 577
Laryngitis, acute.............. 431
  acute subglottic..... 432
  chronic............ 447
  chronic subglottic... 449
  diffuse............. 443
  dry................ 432
  exudative.......... 446
  fibrinous........... 469
  hemorrhagic........ 432
  phlegmonous....... 443
  phlegmonous circumscribed....... 441
  ulcero - membranous 473
Laryngo-fissure.........498, 520, 550
Laryngoscopy, difficult......... 367
  direct.......... 378
  indirect........ 364
  subglottic...... 377
Laryngotomy................. 537
Larynx, anatomy of........... 341
  artificial.............. 552
  cartilage of........... 341

PAGE.
Larynx, catarrh, (see *Laryngitis*) 405
  defects............... 406
  diphtheria............ 469
  exanthemata.......... 467
  extirpation.........497, 552
  foreign bodies...........513
  functions............ 359
  glanders............. 510
  haematoma, traumatic. 522
  haemorrhage.......... 432
  influenza............. 469
  injury............... 521
  joints.........344, 347, 464
  ligaments............ 345
  movements in phonation.............. 358
  movements in respiration............... 359
  movements in swallowing................ 359
  mucous membrane..... 357
  muscles.............. 348
  nerves............... 353
  obliquity............ 405
  paralysis............. 556
  parasites............. 520
  physiology........... 358
  stenosis.............. 408
  tuberculosis.......... 477
  typhoid.............. 475
  vessels............... 355
  whooping-cough....... 468
Lateral pharyngitis, chronic..... 333
Lateral sclerosis, paralysis of larynx in paralysis of soft palate..................... 560
Leech, use of..............113, 520
Leprosy of larynx............ 510
  nose.............. 108
  pharynx........... 316
  throat............. 212
Leptothrix.........109, 215, 228, 317
Leucoma.................190, 208
Leucoplakia, disposition of cancer....................... 190
Levator of palate............. 86
  pad of 238
Lichen ruber.................. 191
Lingua dissectata.............. 178
  geographica 193
Lingual frenulum.............. 181
  mucous membrane..... 172
  muscles.............. 171
  nerves............... 174
  papilla............... 171
  syphilis.............. 207
  tonsil................ 171
  tuberculosis.......... 204
Lip, chancre. See *Primary sclerosis*.

PAGE.

- Lip, cleft, 180
  - cracked, 180
  - fissure, 181
  - leprosy, 212
  - malformation, 180
  - paralysis, 222
  - plaques muqueuses, 208
  - spasm, 223
  - synechia, 181
  - syphilis, 207
  - tumors, 216
- Lipoma, larynx, 530
  - nasal, 119
  - oral, 218
  - pharynx, 321
- Lordosis, 370
- Lorgnette nose, 101
- Ludwig's angina, 227
- Lung affections in laryngeal foreign bodies, 516
  - in stenosis, 414
  - in tuberculosis, 390
- Lupus, 96, 204, 308, 498
- Lymphadenitis, retropharyngeal, 268
  - sublingual, 226
  - submaxillary, 226
- Lymphatic ring, 171, 237
- Lymphoid polypi, 321
- Lymphoma, malignant, 330
- Lympho-sarcoma, 119

- MACROGLOSSIA, 181
- Malformations, larynx, 405
  - nasal, 55
  - oral, 180
  - pharynx, 180
- Malleus. See *Glanders*.
- Massage, larynx, 395
  - nose, 88
  - tonsil, 283
- Measles, larynx, 467
  - nasal, 92
  - oral, 203
  - pharynx, 297
- Meningitis, epidemic, 599
- Microcheilia, 180
- Microglossia, 181
- Mikulicz's disease, 224
- Miliary tuberculosis, 486
- Mineral waters, 249, 281
- Mis-swallowing. See *Disorders of swallowing*.
- Moeller's glossitis, 194
- Mogiphonia, 556
- Mouth and foot disease, 188
  - breathing, 33
  - care, 104, 179
  - cleaning, 179
  - examination, 176

PAGE.

- Mouth, lymphatic glands, 172
  - lymphatic vessels, 173
  - membrane, 172
  - muscles, 171
  - nerves, 174
  - physiology, 174
  - wash, 179
- Mucocele, 134, 147
- Mutation, 361
  - protracted, 589
- Mutism in hysteria, 559
- Myiasis. See *Larvae*.

- NASAL aspiration, 38
  - bones, 4
  - calculi, 109
  - cavity, 1
  - cavity, anatomy of, 1
  - comedones, 50
  - douche, 38
  - elevator, 122
  - inflammation. See *Rhinitis*.
  - injuries, 61
  - polypus, 114
  - polypus, mucous, 76
- Nasal septum, abscess, 62
  - bulging out, 76
  - deviation, 58
  - fracture, 62
  - haematoma, 62
  - naevus, 53
  - perforation, 89
  - perichondritis, 61
  - polypi, bleeding, 114
  - protrusion, 76
  - protrusion backwards, 76
- Nasal wall, lateral, 3
- Naso-pharyngeal catarrh. See *Rhinopharyngitis*.
  - fibroma, 323
  - polypi, 323
- Naso-pharyngeal space adhesions, 257
  - anatomy, 233
  - examination, 24
- Nervous lesions, larynx, 553
  - mouth, 220
  - nose, 121
  - throat, 331
- Neuralgia, laryngeal, 555
- Nigrities, lingual. See *Hair tongue*.
- Noma, 202
- Nose, copper, 46
  - frozen, 51
  - function, 13

PAGE.
Nose, inflammation............ 48
  injury.................. 61
  lorgnette................ 101
  oblique................. 32
  red..................... 46
  saddle................86–105
  stenosis................. 33
  tamponade.............. 66
  vessels.................. 12
  vestibulum.............. 3
Nose-bleeding, free, habitual ... 62
Novocaine.................... 22
Nystagmus of vocal lips........ 589

OBLIQUITY of larynx........... 405
Oedema, angio-neurotic........ 441
  climacteric........... 441
  congestive........... 441
  inflammatory......... 441
  iodide, after use of..... 441
  laryngeal............. 441
  menstrual............ 441
Oidium albicans............113, 212
Olfactometer.................. 16
Olfactory chink-rima........... 12
  membrane........... 12
  nerve.............. 12
  region.............. 12
Operative injuries............. 524
Oral cavity, anatomy.......... 169
  examination....... 176
  function.......... 174
  lymphatic glands.. 172
  lymphatic vessels.. 173
  muscles.......... 171
  nerves........... 174
  physiology........ 174
  vessels........... 173
Orbital abscess in frontal sinus disease..................... 148
Orchitis...................... 225
Oscillation of vocal lips in paralysis....................... 223
Ossification in tonsil........... 237
Osteoma of accessory cavity.... 163
  nose.............. 119
Ostium, front................. 6
  maxillary............. 3, 7
  maxillary accessory.... 7
  sphenoidal........... 10
Overcrossing of arytenoid...... 406
Overstrain of voice.....402, 431, 589
Ozaena. See *Rhinitis Atrophicus.*
  coccus................ 83
  larynx................ 450
  tracheal.............. 450

PACHYDERMIA, laryngeal........ 448
  treatment....... 455

PAGE.
Pad, levator palati............. 238
  Passavant's.............. 240
Palate injuries by operation..... 295
  malformation........... 255
  paralysis.............. 333
  perforation............ 295
Palatine anatomy............. 233
  arches............... 237
  arteries.............. 239
  hook................ 25
  nerves............... 240
  tonsil............... 237
Papillae, circumvallatae........ 171
  foliated.............. 171
  fungiform............ 171
Papillary, excrescences in papilloma...................... 357
Papilloma, laryngeal........... 528
  lips............... 217
  nasal.............. 119
  pharyngeal......... 321
Paraesthesia, laryngeal......... 555
  nasal............ 124
  oral............. 222
  pharyngeal....... 332
Paraffin, frontal sinus.......... 152
  injection of, in rhinitis . 88
  saddle-nose.......... 105
Parageusia.................... 221
Paralysis agitans...........333, 589
Paralysis of abductors.......... 567
  of adductors.......558, 564
  bulbar............... 560
  central.............. 558
  cortical.............. 558
  hysterical............ 558
  in internus........... 566
  inferior...........566, 588
  lateral............... 566
  of lips............... 223
  myopathic........... 556
  neuropathic.......... 556
  posticus..........561, 568
  of soft palate..305, 333, 561
  of tongue............ 223
  transversus.......... 566
  of vagus............. 561
Paraphonia. See *Hoarseness.*
Parasites in accessory cavity ... 164
  in larynx............ 520
  in nose.............. 113
Parchment rattling............ 134
Parosmia..................... 123
Parotid, inflammation of....172, 224
  tumors of............ 229
Parulis...................... 217
Passavant's pad............... 240
Pavor nocturnus in adenoids.... 285
  in pseudocroup 436
Pemphigus, larynx............. 446

PAGE.

- Tabes ataxy of vocal cords 588
  - larynx crises 582
  - paralysis of posticus 333, 568
- Tamponade, Gottstein's 37, 88
  - of nasopharynx 293
  - of nose 66
- Taste examination 175
  - lesions 220
- Tastebuds in larynx 174, 356
  - soft palate 174
  - tongue 174
- Teeth, aberrated 109
- Teleangiectasis of septum 52
- Temperature of foods and beverages 254
  - of room 46
- Thornwaldt's disease 275
- Thymus hypertrophy 412
- Thyrotomy, foreign bodies 520
  - benign tumor of larynx 526
  - cancer of larynx 550
- Timbre of voice 360
- Tin bolts, dilatation 429
- Tobacco 46
- Tongue abscess 184, 215
  - anatomy 169
  - carcinoma 219
  - cysts 216
  - dissectata 178
  - function 174
  - furred 178
  - geographical 193
  - glands 172
  - hair tongue, black 192
  - hyperaesthesia of. See *Glossodynia.*
  - malformation 180
  - mucous membrane 172
  - muscles 171
  - nerves 174
  - paralysis 222
  - spasms 223
  - syphilis 207
  - tuberculosis 204
  - tumors 217
  - vessels 173
- Tonsil, accessory 255
- Tonsil, lingual, hypertrophy 217
  - inflammation 262
  - mycosis 318
  - phlegmon 184
  - syphilis 311
  - varicocele 219
- Tonsil, Luschka's 236
- Tonsil, palatine, abscess 237, 288
  - abscission 295
  - calculi 320
  - carcinoma 327
  - concrements 278

PAGE.

- Tonsil, palatine, diphtheria 300
  - examination 243
  - foreign bodies 318
  - function 240
  - hypertrophy 293
  - inflammation 279
  - lymphoid polypi 321
  - lymphosarcoma 330
  - papilloma 321
  - vicarious bleeding 63
- Tonsil, pharyngeal, abscission 289
  - function 241
  - hypertrophy 284
  - inflammation 260
  - meningitis 599
  - tuberculosis 284
- Tonsil, pendulous 255
- Tonsillitis. See *Angina.*
- Tonsillotomy bleeding 239, 295
  - in singers 596
  - infection 297
- Torus tubarius 277
- Trachea, anatomy of 233
  - bifurcation 358
  - malformations 255
  - mucous membrane 237
  - stenosis 408
- Tracheocele 406
- Tracheoscopy, direct 379
  - indirect 377
  - superior 373
- Tracheotomy, complications of 420
  - diphtheria 474
  - foreign bodies 518
  - laryngeal abscess 446
  - perichondritis 464
  - paralysis 577
  - scleroma 508
  - stenosis 419
  - tuberculosis 498
- Trachoma of vocal cords 449
- Transillumination 30
- Tremolo of voice 592
- Trichina spiralis 521, 557
- Tuba eustachii 233
- Tuberculoma, larynx 486
  - mouth 206
  - nose 96
  - pharynx 310
- Tuberculosis of accessory cavity 165
  - of larynx 477
  - of nose 96
  - of oral cavity 204
  - of throat 477
  - of tongue 205
  - of tonsil 284
- Tumor, accessory cavity 162
  - gum 217
  - intranasal 162

PAGE.

Tumor, larynx 525
  lips 216
  nose 114
  oral 216
  pharynx 321
  salivary 226
  tongue 217
Turbinal bone, med 4
  inf 4
  sup 4
  anatomy 4
  operations on 81
Typhoid, furred tongue 203
  laryngeal 475
  nose bleeding in 92
  pharyngeal 300

ULCER, actinomycosis 114
  aphthous 187
  catarrhal 435
  dentition 201
  foreign bodies 109, 513
  glanders 107, 212, 315, 510
  lenticular 309, 479
  leprous 108
  lupus 96, 204, 498
  mouth and foot disease 188
  noma 202
  parasites 113
  perforation of septum 47, 89
  pharyngeal 274
  tubercular 96, 204, 308, 365
  typhoidal 300, 475
Urticaria, oral cavity 186
  pharynx 273
Uvula, abscission 283
  bifida 255
  cleft in bifida 255
  papilloma 321

PAGE.

Uvula, size 255
Uvulotomy 283

VAGUS 353, 562
Varicellae 186, 204
Varices, larynx 449
  tongue 209
Variola 92, 186, 204, 298, 467
Velum palati 233
Ventricle, Morgagni's 346
Vocal cords 346
Voice 360
  alteration of 387
  breaking of (see *Mutation*) 589
  climacterium 590
  dysmenorrhoea 590
  examination of 363
  exercises 596
  false technique 590
  hygiene of 360
  overstrain 590
  pitch 360
  production 360
  register 361
  senile 590
  singing 589
  timbre 360
  whispering 361
Vomitus matutinus 278

WHISPERING 361
Whooping-cough 202, 468
Wolf's throat 181
Wounds, cut of larynx 522

XANTHOSIS of nasal mucous membrane 85
Xerostomia 178